THE ACUTE MEDICAL SYNDROMES AND EMERGENCIES

Diagnosis and Treatment

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INTRODUCTION

No period in the history of Medicine has witnessed a greater change in scientific and clinical concepts than in the years following World War II This is especially true in the diagnosis and treatment of the acute medical syndromes statistics indicate that many newly developed life saving measures have placed a number of former critical medical emergencies on a more successful level of management The discovery of new drugs new instruments and new procedures have widened the scope of most of the medical syndromes previously considered to be inevitably fatal and hopeless Added to these advancements are the increasing professional implications engendered by the rapid expansion of prepaid medical care projects health and accident insurance and compensation medicine with all of the complicating factors inherent in a third party system

All of these new facets of modern medical practice have focussed attention upon the need for reconsideration and reappraisal of many former methods employed in the diagnosis and treatment of the various acute medical conditions This is not to say however that all previous practices are ready for abandonment on the contrary practical clinical experience has demonstrated that a number of older methods still

serve very useful purposes modified perhaps in some instances by recently acquired knowledge related to their special field of application

The constant challenge of *newness* is faced by every physician the problem of integrating what is new with that which is old the question of accepting the recently discovered and discarding that which is no longer of value and the re evaluation of cherished concepts in the light of scientific progress in the various disciplines are all important factors in the growth and advancement of clinical medicine To the busy physician however the final selection may be confused or delayed by a variety of unrelated issues concerned with time expediency and opportunity at the same *time there is always the personal awareness that sooner or* later some compromise must be made in his traditional concepts as these are faced with newly acquired scientific information

The problem of such selection is admittedly not an easy task and upon those who attempt to render decisions which may be far reaching there rests a burden of responsibility which is unreservedly recognized by the authors of this volume In this connection it should be said that it is doubtful that any single individual regardless of his medical background and achievements would venture alone to cover the wide area included in the consideration of the acute medical syndromes and emergencies in an age of increasing specialization the limitations of specific knowledge and experience makes it impossible for one single author to assume the implied obligations involved in a work of this magnitude and scope

This book is based upon practical experience and full use

of statistical data from all available sources has been employed When the volume was conceived the subject matter was allocated in accordance with the results of a nation wide poll in which physicians in active practice were asked to list the type of important acute medical problems which they had been called upon to treat in the previous 5 years Concerning 3 general groups there was an astonishing agreement these were cardiovascular gastrointestinal and pulmonary in the order named A variety of medical emergency conditions constituted the fourth group the chief of these were acute diabetic problems acute nephritic crises with uremia urinary suppression and edema and finally acute barbiturate poisoning The volume is thus divided into six sections each concerned with these basic specialties of internal medicine It is fully recognized of course that considerable overlapping of these fields of interest may and frequently does occur in certain individuals and cross reference in such instances is freely employed

In attempting to keep the volume as concise as possible and insofar as it has seemed to be expedient detailed presentation of anatomic and physiologic subject matter has been omitted it has been assumed that the reader is familiar with the basic science aspects of the various syndromes discussed On the other hand a free discussion of the common problems involved in diagnosis and treatment permits a broader scope and usefulness of the book

Most of the opinions expressed are the results of the personal experience of the writers and represent their considered judgment in certain critical situations which have issues subject to debate It is in these instances that it is hoped that the volume will serve the physician in those trying hours

which are so frequently a part of the acute medical syndromes In the management of the various emergency conditions where the difference between life and death is spelled out by prompt recognition and treatment of the basic pathology responsible for the situation the authors have spared no effort in the attempt to clarify each problem that may occur

The authors have enjoyed writing this book and wish to extend grateful appreciation to their respective assistants and to their secretaries for their technical help A word of thanks is also given to the publisher Mr Georg Landsberger for his wholehearted support during the preparation of the manuscript

Albert Salisbury Hyman

Part I

THE CARDIOVASCULAR EMERGENCIES

ALBERT SALISBURY HYMAN M D F A C P F A C C D I M

I HEART AND CHEST PAIN

"Heart pain occurs in many forms and in many conditions Perhaps 50% or more of all painful complaints attributed to the heart have no cardiac etiology moreover the point of reference may cover a wide area involving the entire chest and upper abdomen Individuals complaining of heart pain employ a wide variety of descriptive phrases colored by a number of preconceived ideas both true and fancied Pain in the heart in contrast to other types of pain always carries a more ominous significance to many heart pain is synonymous with sudden death It is little wonder therefore that painful sensations arising in or near the precordial area are more likely to be the chief reason of seeking medical care than any other symptom

Many studies have been made of heart pain like all other types of somatic pain the responsiveness factor as well as psychogenic embellishments play an important role in

any given patient's subjective reactions The actual severity of the painful stimulus the specific localization of the point of maximum intensity, its mode of onset its relation to physical or emotional stress its duration the pattern of its subsidence and the post episodal symptoms provide a clinical framework within which the practitioner may quickly separate the pseudo-types of heart pain from those with cardiovascular implications The concept of "pseudo heart pain" requires a few words of explanation the phrase was in wide use over a century and a half ago to explain the phenomenon of precordial pain arising in other organs and structures near the heart "Heart burn for example was originally considered to be an overflow of stomach acid on the heart later in the 1890's stimuli arising in various organs were said to reach the heart by way of the autonomic nervous system Thus a diseased gall bladder could produce painful symptoms referred to the cardiac area In 1932 the concept of pericardial displacement and pressure from adjacent structures received the attention of physiologists and clinicians One of the most common recognized causes of pseudo heart pain is elevation of the left leaf of the diaphragm by a gas-filled stomach eructation of gas is usually promptly followed by lessening or complete disappearance of the "heart pain" in such patients

EVALUATION OF HEART PAIN

Since heart pain is purely a subjective experience the examiner is limited to the information which can be obtained from the patient A comprehensive routine of questioning is necessary and may greatly simplify an otherwise confused and apparently complicated clinical picture During an attack of

severe pain an individual is not likely to take kindly to a long interrogation but a few well chosen questions may secure sufficient data to make a presumptive diagnosis and to start treatment promptly At the very beginning of the questioning it is important to convert the patient s attention from a subjective to an objective viewpoint Parsonnet called this a change from the first to the third person It is a general experience that pain studied in this light usually becomes less dramatic and less fearsome In fact many episodes of pain have entirely or partially subsided during the detailed discussion of the condition with the patient

The actual severity of the pain should be objectively estimated here the dolor scale of Besser is of great value The patient is asked if he can recall the most severe pain that he has ever suffered this may have been a toothache a fracture an attack of acute appendicitis a gall stone episode Using this in a scale from 1 to 10 he is now asked to grade the present pain in relation to the maximum pain which he has remembered In most instances the severity of the pain which was first described as "excruciating" or unbearable" is now reduced to grade 6 or 7 and sometimes to 4 or 5 On the other hand if the pain is still graded as 9 or 10 its intensity can be accepted as clinically significant

Localization Specific localization of the point of maximum intensity may have objective implications Extracardiac causes of "heart pain" usually have a wide distribution which may encompass the entire chest the epigastrium and even the left and right upper quadrants of the abdomen The patient should be asked to place a finger on the most painful spot While true heart pain has no common pattern the in

dividual with angina pectoris for example quickly localizes the area which is most painful In pseudo heart pain the patient may point to 3 or more points widely separated and usually ill defined in repeating the localization a few minutes later new points may be indicated and the previous ones omitted This rarely occurs in true heart pain

It should be pointed out here that the entire subject of heart pain is still in a highly controversial stage the complete mechanism of the excruciating and shock producing pain of coronary occlusion and myocardial infarction is unknown Open surgery of the heart has shown that the myocardium *per se* has few if any pain fibers in contrast to the pericardium and serosa of the root of the aorta Afferent nerve fibers of the baroceptor type apparently carry the pain stimulus both through the vagus and sympathetic plexus systems This absence of clearly defined nerve pathways may be responsible for the confusing lack of correlation between actual cardiac pathology and the development of the pain pattern It is not unusual to note minimal areas of myocardial infarction at post mortem examination in individuals who had suffered extremes of heart pain associated with severe shock the reverse is equally true Many patients dying with massive infarction of the heart muscle experience little or no pain during the entire attack

Referred pain particularly to the left shoulder arm hand and fingers rarely occurs in the extracardiac group Occasionally the referred pain is more severe and lasts longer than the precordial pain Gallbladder disease may produce referred pain in the right shoulder but it rarely produces pain in both the precordium and right shoulder at the same time

Anginal pain may also be referred to the right shoulder but usually it is also associated with left shoulder distribution

The mode of onset of the pain pattern is usually different in the two groups the common stabbing sharp pains which last but a second or two are nearly always extracardiac in origin Like the familiar stitch in the side" the mechanism is probably due to pinching of the diaphragm or a sudden kinking of the splenic flexure True heart pain has a more gradual onset and may not reach its maximum intensity for several minutes anginal pain for example may have a relatively long prodromal period before the painful sensation is actually experienced A long lasting heaviness or pressure sensation over the lower precordium is usually not of cardiac origin many gastrointestinal syndromes exhibit this symptom On the other hand heaviness over the upper end of the sternum after physical effort is not uncommon in aortitis it may precede an anginal episode in certain patients with atheromatous disease with both coronary and aortic involvement Secession of activity is usually followed by a slow subsiding of the painful pressure

Position and posture of the patient sometimes offer a clue concerning the etiological background for heart pain The anginal sufferer usually prefers to stand up or lean over a chair or similar support during an attack he tends to remain in a fixed position This is in contrast with a patient during the initial phase of a painful coronary episode here the individual is restless He may sit down rise quickly walk around the room lie down get up he may groan loudly belch gas become nauseated and finally vomit The anginal sufferer is usually a quiet individual during the painful seizure

PSEUDO HEART PAIN

Patients with pseudo heart pain rarely reach a self estimated dolor scale value of 6 to 7 anginal and coronary subjects reach 8 to 10 The appearance of the two types of heart pain may be characteristic the pseudo heart pain patient may complain bitterly of his pain but he does not look particularly ill True anginal or coronary subjects have the appearance of stricken individuals Brooks once said that the clinical significance of pain in the left chest is frequently in direct proportion to the patient s loquatiousness the more talk the less is likely to be the importance of the symptom Here women are usually more vocal than men but there may be many exceptions to this general rule

Pain due to cardiovascular abnormal physiology is ordinarily of shorter duration than that resulting from extra cardiac causes as for example from a gastrointestinal syndrome except in coronary artery closure with myocardial infarction most episodes of heart pain subside within 10 to 30 minutes Occasionally an attack will last somewhat longer Pseudo heart pain may distress the patient for many hours or days it may be continuously present in a more or less constant degree of intensity for several days During this period the subject may proceed with his customary activities with little or no self imposed restriction physical examination ordinarily reveals no changes in pulse rate blood pressure or ECG alterations With few exceptions myocardial infarction secondary to coronary artery occlusion will show a number of objective findings which will permit a differential diagnosis it is true however and this ob

servation should be emphasized that a small group of coronary patients may experience a more or less major occlusion with minimal subjective complaints not severe enough to force the individual to seek medical care This type of symptom free coronary disease will be considered in greater detail elsewhere but physical examination if permitted frequently reveals a number of telltale findings which are sufficient to render a presumptive diagnosis

THERAPEUTIC APPROACH IN DIFFERENTIAL DIAGNOSIS

It should be pointed out clearly that like all other therapeutic tests in differential diagnosis there may be a wide area of conflicting opinion concerning the interpretation of the results The differential diagnosis of true from pseudo-heart pain is not to be oversimplified by a single test of therapeutic response but in those instances where patient cooperation and intelligence is good the method has much to recommend it The test has other implications hyperacidity is not uncommon in anginal sufferers and in some patients the aluminum gel preparations definitely lessen the need for nitroglycerine It is doubtful however that nitroglycerine produces any beneficial effects in the gastrointestinal disturbances which may simulate true heart pain although a number of authors have pointed out that in certain gall bladder conditions the drug may have some effect in reducing the pain factor

EXERCISE IN DIFFERENTIAL DIAGNOSIS

Exercise and physical activity is definitely contraindicated in true heart pain patients with angina pectoris and coronary

insufficiency will not ordinarily be willing to perform any type of exertion during an episode of heart pain This attitude stands in sharp contrast to the pseudo heart pain group where there may be no self limitation of such exercise It has been emphasized that the anginal subject stops all activities during an attack individuals who experience the sharp stabbing pains of subdiaphragmatic conditions may cease work for a moment but they then continue with their activities Patients with pseudo heart pain do not hesitate for example to climb a flight of stairs during an episode anginal sufferers have learned from experience that such stair climbing is foolhardy

Exercise is thus one of the fundamental differential tests in distinguishing true from pseudo heart pain Physical effort rarely increases the heart pain due to extracardiac causes in some instances the pain may be lessened or even disappear during exercise The psychosomatic implication here is in relation to the well known observation that the subject s attention is easily diverted from his painful sensations Patients of this type frequently say that when my mind is on something else I can forget the pain This rarely occurs in true heart pain which usually has a primary level of importance in the patient s stream of consciousness regardless of what he may be doing

The exercise test must of course be employed with caution common sense and experience will guide the physician in the selection of cases in which the test is likely to be the most promising in differential diagnosis Taken together with other objective evidence the exclusion of the heart and aorta as the cause of the patient s complaints a more definite approach to therapy is made possible

OTHER CAUSES OF HEART PAIN

A word should be said about the etiologic background for heart pain not due to cardiac pathology the gastrointestinal and diaphragmatic group have already been discussed since these are the most common types encountered in clinical practice Many conditions in the thorax may be productive of heart pain 41 such syndromes have been described The most important in the order of frequency are mediastinal tumors both benign and malignant pericarditis aneurysm of the aorta at various sites from the first portion of the arch to the diaphragmatic area many diseases of the lung and bronchi, esophageal pathology orthopedic and neurologic *problems involving the bony structure of the chest and* ver tebrae with associated nerve changes certain disturbances of the sternal bone marrow system and late post traumatic reactions of the chest Herpes zoster and a few rare skin diseases complete the possible list

Differential diagnosis is chiefly based on the discovery or the identification of the abnormality present in the thorax Ordinarily the characteristics of the painful symptoms follow the pattern already described in relation to mode of onset response to nitroglycerine and the effect of exercise of the non cardiac group

It should be repeated here that true heart pain may occur with any of the various conditions described above angina pectoris and coronary artery disease is not uncommon in patients of older age groups who may also be suffering from these pathologic conditions Coronary insufficiency and myo cardial infarction is not uncommon in patients with advanced

pulmonary disease cor pulmonale may be responsible for an hypoxial level leading the anginal syndrome

HIATUS HERNIA AND HEART PAIN

Hiatus hernia deserves a special section in its relation to the production of heart pain With more accurate methods of diagnosis this syndrome is becoming one of increasing importance It has been demonstrated by a number of observers that hiatus hernia may mimic many of the clinical manifestations of coronary heart disease This may be due to the fact that in certain individuals the same vagal and sympathetic pathways are intimately associated in the neurologic network supplying both the heart and lower end of the esophagus and the cardiac portion of the stomach A disturbance at the lower level of the plexus is reflected in the cardio aortic area It is not uncommon to note premature beats changes in cardiac rate and blood pressure during an acute hiatus hernia episode More important from a clinical standpoint insofar as diagnosis is concerned are the changes which may occur in the ECG the depressions of the ST segments and inversions of the T waves in significant leads together with the type and distribution of the heart pain readily suggest coronary occlusion with myocardial infarction

In most instances these ECG abnormalities clear up within a few hours or days there is usually no associated laboratory response WBC sedimentation rate and transaminase levels remain unchanged There may however be a delay in restoration of the ECG pattern and some or all of the laboratory data may be suggestive of a true coronary episode Here the differential diagnosis is exceedingly difficult and the physician has no choice under the present methods of exam

mation except to consider the situation as one of presump tive coronary closure and he must plan his therapeutic procedures accordingly The author has seen 2 such hearts at post mortem deaths were from other causes No objective evidence of coronary pathology was found On the other hand he has also seen 11 instances where demonstrable myocardial infarction has occurred in patients with associ ated hiatus hernia

Hiatus hernia with its associated heart pain and ECG changes thus constitutes one of the most difficult clinical situations which the practitioner will encounter The prob lem involves not only the differential diagnosis but the en tire management of the patient s life and activities the question of whether he has or has not coronary heart disease in addition to his hiatus hernia syndrome will have far reaching implications

ACUTE PULMONARY EMBOLISM

More complicated are the cardiac masquerades of acute pul monary embolism and pulmonary artery occlusion While the clinical aspects of pulmonary embolism are discussed in detail in another chapter the syndrome is considered at this point in relation to heart pain In the overwhelming shock produced by large emboli with complete occlusion of a major pulmonary artery and infarction of lobular areas of the lung little or no heart pain is ordinarily produced Death usually occurs within minutes the patient becomes cyanotic exhibits the delirium of agonal air hunger and dies during a paroxysm of blood streaked coughing Death is probably due to com plete cardiac arrest secondary to an explosive vagal stimulus

ventricular fibrillation as the result of left ventricular collapse has been noted in experimental animals

Smaller emboli which plug secondary branches of the pulmonary artery tree have a mortality rate of about 50% depending upon the site of the closure degree of shock compensation of the right ventricle extent of the lung bed involved and finally the responsiveness of the entire heart to the sudden hypoxial state in which the dangerous arrhythmias may develop In this condition heart pain may be very severe and in the same order of magnitude as acute coronary occlusion with myocardial infarction The ECG changes are characteristic and occur within a few minutes in contrast to myocardial infarction which may require a number of days before the full pattern is developed Pulmonary edema usually occurs rapidly with blood streaked frothy sputum as a rule congestive failure in coronary occlusion develops after several hours or days and hemoptysis is not common The x ray examination of the lungs is rapidly positive with characteristic changes films taken within 15 minutes of the onset of the attack may be diagnostic the lung findings in coronary occlusion are negative even though considerable congestive failure is noted by auscultation

Differential diagnosis is important in the consideration of emergency treatment pulmonary embolism primarily involves failure of the right heart while coronary occlusion involves the left ventricle Venous pressure may be considerably elevated in pulmonary embolism and venous decompression should be performed promptly an initial withdrawal of 500 cc is usually indicated The blood should be handled carefully as it may be used later for replacement transfusion Pain should be controlled by Demerol or similar narcotics

hypoxia should be treated by adequate oxygen therapy Quinidine or Pronestyl can be used if arrhythmia develops digitalis may do more harm than good in the acute phase of pulmonary embolism

Convalescence in successfully treated cases of pulmonary embolism is usually rapid in contrast to the long disability in acute coronary occlusion Depending upon the severity of the pulmonary reaction ambulation may be permitted in a week or 10 days The source of the embolus will have some bearing upon subsequent treatment

PSYCHOSOMATIC ASPECTS OF HEART PAIN

Before concluding this discussion on heart pain some consideration must be given to the general reaction of patients to pain in the cardiac region regardless of the mechanism of its development An individual suffering from pain in the heart may only have a superficial academic interest in the abnormal physiology responsible for his disability His painful symptomatology is a highly personal matter which has a superior order of priority in his private life His entire philosophy of living and working may be and frequently is geared to the pain in his chest It has already been pointed out that painful and disagreeable stimuli in this area usually carry a far more sinister significance than such stimuli in any other part of the body The physician in these circumstances is confronted with a double problem differential diagnosis and relief of the patient s complaints

Here the Art of Medicine vies with Science and here the Art of Medicine is interpreted in terms of a psychosomatic approach to the individual s problems The agonizing heart ache of grief depression and despondency may be equally as

real and objective as pain due to cardiovascular pathology there may be a distinct difference however Whereas pain due to heart disease is usually responsive to appropriate therapy the relief of extracardiac pain whether referred or fancied is oftentimes far more difficult to obtain An understanding and sympathetic approach to the patient s complaints may spell the difference between success and failure in many instances An indifferent and cursory explanation that the given pain is not due to heart disease may leave the individual totally unimpressed the thought that the physician is minimizing a dangerous condition by a play on light words is constantly in such a patient s mind An opposite and unintended reaction may actually occur with a worsening of the entire clinical picture

II ANGINA PECTORIS

The basic physiologic concepts concerned in the mechanism of angina pectoris have not changed much during the past 25 years the relationship of the syndrome to coronary artery insufficiency occlusion and myocardial infarction still remains to be more clearly defined within the framework of the stenocardial postulate of the early German authors Recent cardio pulmonary studies have confirmed the generally held impression that localized areas of myocardial hypoxia may be chiefly responsible for the development of the painful and distressing clinical picture While the factors of blood supply and demand still play an important part in the overall disturbance of myocardial metabolism the neurogenic aspects are receiving more research attention Open cardiac surgery and experimental animal studies have shown that simple occlusion of a coronary artery channel with its secondary infarction of the heart muscle segment supplied by the vessel does not necessarily produce the typical pain pattern seen in clinical angina pectoris One school of investigators has placed the origin of the pain mechanism at a higher level than the area of myocardial ischemia this may explain the more or less symptom free types of acute coro

nary artery occlusion with myocardial infarction which occur in certain patients It may also explain to some extent the *modus operandi* of the preventive therapeutic measures which are extremely successful in some individuals

ANGINA PECTORIS AND CORONARY ARTERY DISEASE

A number of cardiologists have fostered the concept that all anginal subjects have a background of coronary artery disease factually this is untrue Post mortem examination of anginal sufferers dying from causes other than cardiovascular frequently show no changes in the coronary system nor the scars of old myocardial infarcts From a clinical standpoint many anginal subjects never present abnormal electrocardiograms even during an attack they may also show no objective evidence of heart disease Such patients may exhibit small hearts by x ray study normal blood pressure and more or less normal functional capacity tests Coronary artery disease is a syndrome of progressive pathology but notwithstanding objective changes in the ECG increase in heart size and loss of functional capacity many patients do not experience anginal seizures If the coronary insufficiency concept of anginal pain were valid these individuals should have increasing episodes The fact is that many patients who have suffered a series of attacks may become entirely free from the syndrome as their coronary disability increases over a period of months or years

Angina pectoris may exhibit a sporadic time factor it is not uncommon for an individual to experience a number of attacks over several weeks or months followed by complete relief for relatively long periods perhaps 3 to 5 years before

reappearance of the syndrome Other patients may have several bouts of angina some of frightening severity and then live a symptom free existence for the remainder of their productive years A case in point is that of a schoolteacher who had a number of attacks at the age of 42 his cardio vascular examination at the time was essentially normal and remained so until at the age of 78 he developed prostatic obstruction Following a difficult anesthesia during surgery he had a period of congestive failure secondary to pulmonary complications and died about 4 months later

MECHANISM OF THE ANGINAL SYNDROME

The *trigger mechanism* responsible for an attack of angina pectoris will be discussed subsequently in the section on pre vention of the syndrome but a hasty appraisal of the *modus operandi* of the episode for which the practitioner has been called to treat may have some significance more especially if the subject has not been seen before If for example the patient developed the attack while performing a certain task or under a given environment attempt should be made to change the principal factors of the situation It might seem unnecessary to make such a statement but many anginal sufferers in their initial episodes have the feeling that they can work off an attack This concept is common in the garment industry among bakers and also with house painters and decorators among the latter as with other individuals who may be working in an atmosphere heavily loaded with industrial fumes and gases removal to a more normal en vironment is important regardless of the patient s unwilling ness to make the change for one reason or another

toothache in an edentulous subject this observation has also been noted by many others The intense pain in a non existent tooth or teeth has been compared to the phantom pain in the foot of an amputated leg in 1926 Buerger and the author described 3 such cases who suffered from thromboangiitis obliterans and angina pectoris

Response to nitroglycerine may show unusual and bizarre pain relief patterns ordinarily the precordial and referred pain zones subside more or less simultaneously but there may be considerable lag in one or another of the areas There may be a tendency in certain patients for the referred pain to persist in somewhat lessened intensity for 10 to 15 minutes tingling and numbness in the hands and fingers for some time after relief of the original attack is not uncommon Some individuals may experience an uncomfortable feeling which is difficult to describe in the shoulder and arm for some hours after an attack It is possible that the *shoulder arm syndrome* suffered by many cardiac patients without angina pectoris may have a common etiologic background insofar as the referred pain mechanism is concerned

ANGINA PECTORIS DECUBITUS

Since the anginal syndrome is primarily the result of a stress mechanism the functional stenocardia occurring at rest has been explained by the postulate of *adynamic hypoxia* While asleep and with lowered basal metabolism the factors of blood velocity blood pressure pulse rate and per minute volume output of the heart are at their lowest level In subjects with a critical marginal reserve the increase in oxygen debt may be productive of the same response as that de

veloped by exertional hypoxia This concept has been challenged by the work of recent investigators like Bishop Sussman Michelson and others

These cardiologists believe that angina pectoris decubitus is due chiefly to autonomic simulation of physical stress while lying in bed asleep a patient may develop an attack as severe and as intense as that following a given type of physical or emotional effort In most instances the trigger mechanism is a recalled or unremembered dream The content of the dream sequence usually includes an activity known to produce an episode a typical illustration is that of a well known golf champion who was forced to give up the game at the age of 54 because of severe attacks while playing The physical effort of the game plus the emotional stress involved in national competition had resulted in complete retirement from playing He remained free from anginal episodes for about 6 months he then began to have attacks at night while in bed He would awaken with symptoms identical with those which he had previously experienced while playing On careful questioning the patient finally revealed that the attacks followed a vivid dream sequence in which he was again playing an intense game in challenge competition this dream content was repeated several times a week and on each occasion he awoke with pain It is well known that children and young adults may experience such vivid dream sequences that they awake with palpitation breathlessness and sometimes precordial pain the autonomic nervous system in such instances is apparently unable to distinguish between actual and dream content activity Meiers in a recent psychiatric study of patients with noc

turnal angina pectoris has found that most of these individuals have a history of childhood or adolescent vivid dream responsiveness which is carried over into later life

The concept of adynamic hypoxia as the cause of decubitus angina was also challenged by physiologists like Opitz and Williams with lessening of the various factors of circulation which occur at rest in bed a common denominator is experienced by all subjects with coronary insufficiency In any given patient episodes of anginal pain should occur every night but factually this is untrue many patients with severe daily bouts of angina never have any episodes at night Likewise individuals with advanced coronary artery and myocardial disease who have frequent episodes of heart pain rarely suffer such attacks while asleep at night Other factors at present unknown or poorly understood play an important part in the chain reaction leading to angina pectoris decubitus

TREATMENT OF THE ACUTE ANGINAL SYNDROME

Nitroglycerine This still remains the drug of choice in the symptomatic relief of the acute anginal syndrome there is no place here for the employment of any of the long list of recently introduced nitrate remedies which have been developed by the pharmaceutic industry as substitutes for nitroglycerine although most of these have a usefulness in the prevention of attacks These will be discussed later in this chapter Nitroglycerine like digitalis and morphine continues to be one of the God given drugs

Nitroglycerine glyceryl trinitrate glonoin in either liquid or tablet vehicle has traditionally been the most consistently effective drug in the management of the pain component

It is promptly absorbed from the mucous membranes of the mouth and is quick acting while sublingual administration is the common method of employment many patients secure relief from buccal absorption as well as from the tip of the tongue The drug has diminished effects when swallowed but there is clinical evidence that it may be quickly utilized when given by rectum in selected cases special spirits of glonoin suppositories are available for this purpose

The full employment of nitroglycerine is frequently mediated by a number of its side effects these may be so severe and disagreeable that patients may refuse to use the drug feeling that the pain of the attack is the lesser of two evils The chief of these subjective effects are the cerebral reactions a severe throbbing headache with flushing of the face and neck is not uncommon Occasionally vertigo and nausea occur but rarely vomiting abdominal symptoms have also been experienced in older patients These reactions are more likely to develop during the initial employment of the drug with continuing use the side effects become less disturbing although many anginal sufferers may experience subjective reactions in a mild form over a long period

The usual preliminary dosage of nitroglycerine is 1/100 of a grain but where the cerebral reactions are a problem one half to one quarter of the tablet may be given about 2 to 5 minutes apart if the anginal pain has not subsided It requires some dexterity to divide the ordinary tiny nitroglycerine sublingual tablet but it may be dissolved in a small quantity of water (about 4 teaspoonfuls) and one teaspoonful at a time may be held in the mouth Whiskey or brandy held under the tongue may be useful in elderly men who are unable to use nitroglycerine even in small doses

Tolerance to nitroglycerine is quickly established in certain individuals where one tablet previously resulted in complete relief 2 to 5 or more tablets may be required after several months or years of employment It should be pointed out here that increased dosage to obtain a given effect does not necessarily indicate that the patient s condition is worsening the tolerance factors must first be taken into consideration The need for increased dosage may also stem from the natural deterioration of the drug which is a highly volatile substance simple exposure to heat and light may cause marked loss in potency Experiments at the Valley Forge Heart Institute and Research Center have shown that when a glass vial containing the standard tablets is kept at *body temperature for 2 weeks nearly 22% of the drug is lost* a similar vial placed in the glove compartment of an automobile during the summer months lost 54% in 18 days Patients should be instructed to divide the usual 100 tablet container into 2 parts 10 to 12 tablets should be carried on the person while the remainder is best kept at the bottom of the home refrigerator This recommendation can also be applied with equal profit to all drugs of the nitrate and aminophylline series

Nitroglycerine is also soluble in certain oils of the edible type "Concentrate A of the Valley Forge Institute formulary contains Vitamin A in a bland oil mixture with glonoin One drop placed on the tip of the tongue with a small glass rod applicator affixed to the stopper of a 10 cc container has given prompt relief to hundreds of anginal sufferers The work of Plungian Wolffe Munch, and others have shown that Vitamin A seems to potentiate nitroglycerine by aiding its rapid absorption from the mucous membranes of the

mouth Nitroglycerine is also potentiated by the sorbitol compounds contained in a gelatine capsule which may be crushed in the mouth the drug is quickly absorbed by the buccal membranes

The Volatile Nitrate and Nitrite Drugs These have long been used in emergency treatment of heart pain amyl nitrite is perhaps the oldest and best known of the group It is usually administered by inhalation of the substance which may be conveniently carried in a glass ampule and crushed in a handkerchief it has however a penetrating and distinctive odor which may be objectionable to some individuals but in others there may be a favorable and specific psychogenic effect from the smell alone Amyl nitrite and ether in equal parts was a favorite remedy in Europe after its introduction by the Viennese cardiologist Kaufmann in 1920 while in the Vachez Clinic in Paris amyl nitrite was combined with an aromatic amoniacal solution and administered from an atomizer bottle Old fashioned "smelling salts were in wide vogue in New York in the 1900 s for the relief of anginal symptoms All of these older inhalation remedies still have a place in selected patients the fact that they are old and nearly forgotten methods does not necessarily indicate that they should not be used In a given patient who has not re sponded for one reason or another to the more recently introduced drug preparations some of these previous sub stances may be of surprising effectiveness

Oxygen The inhalation of 100% oxygen may in certain pa tients give prompt relief the small gas cartridge with a special nasal appliance may be carried in the pocket or purse

Available cartridges are about the size of a cigar and contain 5 liters of condensed gas it may be used for several attacks before recharging

It should be repeated here that every product used for the relief of the acute anginal syndrome carries with it a large and imponderable factor of psychogenic responsiveness This factor may outweigh all other considerations in the patient s evaluation of a given drug

Other Remedies The pain of anginal episodes is sometimes relieved by hot drinks the liquid should be as hot as the individual can tolerate and should be kept in the mouth for at least a minute before swallowing The therapeutic factor here is apparently the heat itself but many individuals favor one drink or another hot tea with lemon, peppermint water black coffee and concentrated salines like magnesium sulphate or sodium phosphate (Fleet) have been employed with satisfactory results It may be pointed out here that since angina pectoris is a very old disease it is natural that its treatment is more or less burdened by a considerable folklore when a given patient is not relieved by medication prescribed by his physician he lends a willing ear to suggestions regardless of the source Under the circumstances the physician must not withhold a sympathetic understanding of the individual s instinctive desire to secure relief from his painful syndrome but he should be ready to offer alternate methods of treatment recognizing that no two patients respond in identical manner to any given routine

PREVENTION OF THE ANGINAL SYNDROME

Treatment of chronic and repeated attacks of angina pectoris is largely based upon prevention of the episodes the chain

reaction leading to the trigger mechanism has already been described The factors of physical effort and strain emotional stress and anxiety visceral and other autonomic stimuli all play a varying role in the final development of any given attack Perhaps in no other disability is a well considered philosophy and planned way of life more important for the immediate and future welfare of the individual

Prevention of angina pectoris thus starts with a careful analysis of all the possible elements which have participated in the chain reaction this may require more time and attention than the physician may feel that he is expected or can afford to allot to a given patient In a busy general practice such a *de facto* analysis and evaluation may however be spread over several visits the cooperation of the patient and his family in keeping a diary or record of the data associated with the episodes is always helpful A prepared questionnaire containing significant items may be given to the individual a number of these are available and will save the physician no little time and energy in obtaining essential information Running throughout all such records a number of pertinent facts usually stand out cause and effect may be closely related but hidden by extraneous observations Simple elimination and emphasis upon corrective measures may in some instances produce gratifying results

DRUG THERAPY

For the prevention of the anginal syndrome drug therapy has in the past decade occupied the attention of increasing numbers of research pharmacologists the agonizing pain of the syndrome as well as the mechanism responsible for it has proved to be a challenging and rewarding field in the

pharmaceutic industry This may stem from the remarkable advance made in the development of new drugs for the relief of pain in general The 1958 list of such preparations contains over 60 new substances unknown only a few years ago These include synthetic drugs of the opium series isomers of the barbiturates and the various tranquilizer preparations The concept of coronary artery dilation as a method of preventing the stenocardial factors of the syndrome has led to the exploitation of various preparations containing many old and some new drugs for this purpose All together there are available more than 100 different remedies which the practitioner may employ

Nitroglycerine Preparations Standard U S P nitroglycerine in 1/100 grain sublingual tablets should always be used in new patients to test therapeutic effectiveness as well as to determine the patient s vasomotor reactions Where larger doses are indicated 1/50 or 1/25 grain tablets are usually more satisfactory than 2 or 4 tablets of standard potency the psychogenic implications are clear in some patients

If repeated doses are necessary because of frequent attacks during a given day or if continued physical or emotional stress is anticipated nitroglycerine in delayed action form may be useful Nitroglyn consists of coated granules of the drug which have a different solubility time factor

Double action tablets which are coated with nitroglycerine for sublingual absorption and which have a base tablet of pentaerythritol tetranitrate that may be swallowed later are available as Dilcoron A combination of these 2 drugs is also *obtainable as* Peritrate with Nitroglycerine (Warner Chilcott)

Nitrate Preparations The nitrate drugs of the pentaerythritol tetranitrate series have long been employed in the prevention and treatment of the stenocardial syndromes although much academic controversy still continues concerning the spastic contraction concept with reference to the coronary system many patients receive symptomatic relief from continued use of these preparations Recent comparative studies have shown that anginal sufferers with minimal objective evidence of coronary or myocardial pathology are generally benefited by these substances the findings here are confirmatory of the postulate of Sussman that rigid or diseased arteries can neither contract nor dilate The number of such nitrate compounds is large and the physician has a wide choice of preparations each of which under a given set of circumstances may be the specific remedy for the individual s complaints

Peritrate (Warner) Corovas (Amfre Grant) Metamine (Leeming) Pentritol (Evron) Nitretamine (Squibb) are all reliable products of this group

In recognition of the psychogenic factors associated with angina pectoris in many patients the tranquilizers have been combined with pentaerythritol tetranitrate Miltrate (Wallace) and Equanitrate (Wyeth) are representative of this series

In a syndrome as protean as angina pectoris it is to be anticipated that drugs employed in the treatment of other conditions may also be effective in some measure in the management of this disability more especially if the other disease is related to stenocardia Hypertension is not uncommon in the anginal syndrome and the useful drugs for this condition may act as a suppressive agent Thus the

rauwolfia preparations alone or in combination with sedatives have a place Theominal R S (Winthrop) and Theso date R S (Brewer) are useful in this connection Rescinnamine an ester of methyl resperate has also been effective The quinine and reserpine combinations have been used

A number of other drugs have been employed under special occasions Ipromiazid for example has shown value in elderly patients Isopropamine Iodide which is an anticholinergic compound may control the pain factor in subjects with a provocative gall bladder Noscapine, one of the isoquinoline alkaloids of opium which has a papaverine like action on the coronary arteries shows promise as a potent drug for angina

Aminophylline Preparations When angina pectoris occurs in patients with more or less advanced coronary artery disease and particularly where pain is a continuing disability after myocardial infarction the theophylline theobromine group of drugs may have a special place in the physician s therapeutic armamentarium When given in large enough dosage to produce optimum cardiac response there may be considerable gastrointestinal reaction heart burn nausea and anorexia is common If the drugs can be tolerated however they may yield satisfactory results over months and years as a group they are the most dependable and consistent in their suppressive effect

Pure aminophylline (Dubin) (Squibb) and (B W & Co) should be given in 250 mg tablets 3 to 6 times a day and may be followed by an alkaline water like Kalak or Celestine Vichy

Some aminophylline preparations contain an antacid or

buffered substance like aluminum hydroxide gel of these Aminodrox Forte (Massengill) and Cardalin (Irwin Neisler) are most useful

For individuals who prefer liquid instead of tablet administration for one reason or another Elixophylline (Sherman) is a palatable alcoholic mixture which is best taken before meals

Rectal administration of aminophylline may be necessary in patients with nocturnal bouts of angina Aminet Suppositories (Ames) and Aminophylline Suppositories (Wyeth) are suggested Clysmathane (Fleet) is a liquid solution of theophylline monoethanolamine (0 625 gm) in a disposable rectal unit which may be employed in emergency conditions associated also with acute pulmonary edema

Again recognizing the need for sedation in angina pectoris a number of aminophylline preparations are available with combined barbiturates of these Choledyl (Nepera) Glytheonate (Patch) Calpurate (Maltbie) Diurbital (Amfre Grant) Theocalcin (Bilhuber) Theominal (Winthrop) Thesodate (Brewer) and Piperophylline (Smith) may be mentioned All are satisfactory and produce consistently good results in certain patients

Sedation and Tranquilizers It has been mentioned several times that the prevention of anginal attacks may in no little part depend upon adequate suppression of those psychogenic factors which are responsible for the development of the chain reaction mechanism The experience of many years has proven the value of moderate sedation in a vast majority of anginal sufferers Here the barbiturate group of drugs has enjoyed wide popularity notwithstanding the danger of

habituation since the physician is offered a wide selection it may be advisable to keep changing any given preparation after a month s continuous administration

Phenobarbital (B W & Co) Nembutal Sodium (Abbott), Barbital (Lilly) Carbrital (Parke Davis) Butisol Sodium (McNeil) Eskabarb (S K & F) Sedobarb (Robins) and Luminal (Winthrop) are but a few of the more common preparations available

Many patients however when the barbiturates are employed in sufficient dosage to produce suppression of the psychogenic factors in angina pectoris may have a tendency to experience a *hangover effect* The drowsiness and more or less languid feeling with its drop or dulling of mental acuity may have significant implications where alertness is important in any given type of work or activity and some individuals while benefiting from lessened incidence of attacks object to the side effects of the drug

Here the tranquilizers are a valuable addition in suppressive therapy they may produce a satisfactory level of suppressive activity by lowering psychogenic responsiveness to the ordinary daily stress and strain of certain patients without depression of cerebral function Miltown or Meprospan (Wallace) Equanil (Wyeth) Thorazine and Compazine (S K & F) and Pathibamate (Lederle) may be mentioned

The Sex Steroids A word should be said about the sex hormones in the prevention of angina pectoris The work of Wolffe Heubener Katz Kurzrock and others have shown that the sex steroids may have a role in dysfunction of cholesterol metabolism and insofar as certain types of stenocardia may be produced by abnormal changes in steroid

chemistry the employment of these substances may be useful in the management of the syndrome when other measures have failed

There is unfortunately no general agreement among cardiologists concerning the method of administering these drugs and in the long range clinical results obtained Nor are the indications for their use clearly defined There are however a number of favorable reports and in carefully selected subjects these substances have proven clinical value In our experience men have benefited far more than women the men more likely to show favorable results are those with obvious signs of the male climacteric and with a high emotional background Here the various androgen preparations in clinical dosage over a prolonged period of months and sometimes years has proved to be a valuable supplementary therapeutic measure In some instances combined androgen estrogen medication has been more effective than when the androgens have been given alone Brilliant results have been noted in men of the 50 to 60 year group with hypertension and certain autonomic nervous system syndromes like spastic colon gastric hypermotility with and without ulcer complications and sinus tachycardia This is the more or less familiar picture of the high strung overworked business executive or professional performer who among other personal problems recognizes an increasing sexual impotency irritability in small matters insomnia loss of appetite and perhaps a decrease in mental capacity Angina pectoris is a common disability in this group and the sex hormones should be given a clinical trial with due attention to the prostatic implications

When the anginal syndrome develops in women during

the menopause the results of estrogenic medication of various types have not been particularly striking and actual appraisal has been difficult however in certain specific instances it has appeared that the estrogens may have potentiated other remedies so that the patient has received some benefit The mixed androgen estrogen preparations have not been useful in women

SURGICAL TREATMENT OF ANGINA PECTORIS

In any large series of anginal subjects a relatively small group averaging from 5 to 8% remain resistant to all medical procedures as a rule these patients exhibit extensive cardiovascular pathology Hypertension cardiac hypertrophy aortic disease abnormal ECGs are common findings occasionally there is evidence of congestive failure with minimal edema Ordinarily there is little or no response to nitroglycerine or to any of the other drugs employed in the management of stenocardial symptoms Such patients may present the typical picture of the anginal syndrome with cause and effect easily established but more often the attacks of pain are apparently unrelated to disturbances in functional capacity Pain in various degrees of intensity may be present continuously

Although the name *status anginosis* has been applied to this syndrome it is doubtful that this condition has the same pathologic background or is due to the same physiologic mechanism as the nitroglycerine responsive type of angina pectoris Clinical differences can be noted in the two groups the classical referred pain pattern is usually absent in status anginosis The paroxysm of pain is less severe in the latter

group but the patient has the appearance of an individual in mild cardiogenic shock

The Surgical Approach To the management of such difficult cases surgery leaves much to be desired notwithstanding the great number of reported cures and improvement in the patient s general condition A number of procedures have been employed thyroidectomy was a common surgical con cept in the 1930 s With the advent of radioisotopes I131 has taken the place of actual removal of the gland In patients with intractable congestive failure and heart pain the overall results have been moderately successful insofar as improve ment in symptoms are concerned but life expectancy has not changed materially

Paravertebral Alcohol Block For the relief of the pain factor in hands of competent surgeons or local anesthetists this has been the method of choice in some cases the relief from pain however may be rather short In 6 instances of the author s series the block procedure had to be repeated within 3 to 4 months Two patients had 5 such treatments within 2 years Relief of the pain factor has produced a number of controversial questions Opitz and Cannon pointed out that from a physiologic aspect heart pain is an autonomic signal of danger to the organism Like a red light to a motorist it calls on the individual to stop if the signal is missing or ignored the danger of accident is increased Without the warning symptom the anginal sufferer is likely to continue with his activities with increasing hypoxia if the locus of minimal resistance to ischemia is in the myocardium the pos

sibility of infarction is enhanced On the other hand such myocardial injury is not frequent in post anesthetic block cases most of these patients have extensive coronary disease where such episodes are not unexpected

Other Types of Surgery Promise in the relief of the syndrome is shown by other types of surgery the chief of these is the attempt to secure a better blood supply to the heart muscle The least difficult to perform is artificial pericarditis Thompson Bailey Glover Bloomberg and other cardiac surgeons perform this procedure with minimal reaction to the patient and with subsequent improvement in varying degrees insofar as symptomatology is concerned Ordinarily there is little or no increase in functional capacity although some patients are able to increase their physical activities

More Complicated Surgery Ligation of the coronary veins and the introduction of new arterial connections with the myocardium by transplant from other adjacent structures like the internal mammary artery, involves more complicated surgery In the hands of experienced operators and in selected cases the outlook has definitely improved in the last decade New techniques new types of materials new methods of open surgery hypothermic anesthesia have all produced consistently better overall results and the physician should be familiar with the advantages of surgery in patients who fail to respond to medical treatment Cardiac surgery will have an increasing role in the management of patients with advanced heart disease in the correction of valvular defects the direct approach has proved to be an accepted

method of treatment In the relief of the stenocardial syndromes and in chronic intractable congestive failure the results have been less conclusive but must be considered in any given case where other methods have failed

III CORONARY ARTERY OCCLUSION AND MYOCARDIAL INFARCTION

The clinical syndrome of acute coronary artery occlusion with its associated myocardial infarction is pathologically one of localized irreversible hypoxia The physiologic point at which the phenomenon of hypoxia becomes irreversible may be difficult to determine or demonstrate but is at this level of oxygen debt that simple angina pectoris becomes a manifestation of organic or structural coronary insufficiency In the previous chapter it was emphasized that many patients may suffer from repeated attacks of angina with no objective evidence of heart disease even post mortem studies have failed to reveal counterpart pathologic changes in the cardiovascular system The mechanism was postulated to be due to a functional type of stenocardia with a temporary phase of ischemic hypoxia of the heart muscle When the functional capacity of the coronary system is diminished by any disease process which limits arterial flow and when the balance between supply and demand is inadequate under a given condition a similar but irreversible symptom complex may develop

The Hydraulic or Hemodynamic Factors In coronary artery disease these factors have a major role in functional capacity

of the vascular network The chief type of pathology responsible for narrowing the lumen is atheromatous disease involving the intima which becomes thickened and swollen ✓ Here the simple hydraulic laws of Preistly become operative in 1688 he proved that a 2 inch wooden pipe carried 4 times as much water as a one inch pipe The reverse is equally true reducing the internal diameter of any tube by one half will reduce its capacity to one fourth From a clinical standpoint any change in the size of a vessel lumen will considerably reduce its functional capacity In thrombus formation there is thus a marked restriction in possible blood flow in accordance with this hydraulic law

A more complex hemodynamic factor is related to the pressure gradient changes required to deliver a given volume of blood through a narrowed lumen in a certain period of time The time factor is important in the demand and supply balance upon which the syndrome of coronary insufficiency is based If the immediate volume of blood is inadequate to meet the demands of myocardial metabolism an early phase of hypoxia develops this phase may be changed if supply quickly compensates for the ischemic alterations When on the other hand there is a delay in either the necessary adjustments in volume or pressure significant disturbances in heart muscle metabolism occurs which may eventuate in death of the tissues involved

Infarction of a Heart Muscle Segment This may occur without actual occlusion of a given coronary artery if the vessel has a functional end artery anatomic relationship to a certain myocardial area increased metabolic demands of the muscle segment may exceed the carrying capacity of the artery

This is not uncommon in the subpericardial and subendocardial layers of the heart muscle where single and multiple small infarctions may be noted in the presence of more or less normal proximal coronary arteries

Complete occlusion of the smaller subdivisions of the coronary tree may result from emboli released from thrombotic masses which have developed in the major branches the smaller vessel may have been entirely normal before the fragmentation process Such emboli are likely to be multiple with several areas of infarction arising at the same time or in a sequential time pattern in which the subject has 2 or more clinical attacks during the first few days or a week after the initial episode

While there is no consistent factual data in regard to the mortality statistics in relation to embolic compared to thrombotic occlusion of the coronary arteries our experience suggests that the embolic type of myocardial infarction has a much better prognosis In certain instances this may have been due to the greater areas of infarction involved in thrombosis of the larger vessels but in some autopsy specimens the damaged areas have been no more extensive than in the embolic group

DIFFERENTIAL DIAGNOSIS FROM ANGINA PECTORIS

It may be impossible to distinguish severe episodes of angina pectoris from acute coronary artery occlusion As a general rule it may be said that if an attack of angina is not relieved within 20 to 30 minutes, occlusion must be suspected and the patient treated accordingly This is particularly true if the individual has experienced a number of previous anginal attacks all of which have responded for example to nitro-

glycerine There is some evidence to indicate that every *serious* attack of angina pectoris must be regarded as an actual if not a potential coronary episode *serious* is here interpreted to be unusual in some manner from the ordinary and customary attacks which the patient has previously experienced The pain may be *more intense* have a wider radiation pattern last much longer after the employment of the habitual remedies and is accompanied by symptoms not noted before These may be dyspnea palpitation sweating weakness prostration or even collapse

There are a number of distinguishing characteristics which may be recognized in the typical instance of acute coronary artery closure the patient shows a grayish distinctive pallor in contrast to the flushed facies of anginal subjects The individual has a tendency to sit crouched in a chair or he may walk agitatedly up and down the room while an anginal sufferer usually remains standing in fixed position In coronary episodes the patient moans with pain he is talkative and tends to be *noisy* while an anginal subject is usually *quiet* Cold clammy sweating of the face neck, and chest is common in the coronary group this is unusual in angina pectoris

Functional Hypotension The rule in coronary closure is functional hypotension it may be elevated during an anginal attack Some authors have noted a rapid pulse in the former and no change in the latter but in our experience changes in pulse rate have not been consistent in either group The same may be said about the heart sounds while it is true that the sounds become fainter and more distant in the coronary group and louder in the anginal cases there may

be many variations at the very onset of the attack *Failure of the heart sounds* is more common after the first several hours in occlusion but many patients pass through the entire first and second stages of myocardial infarction with relatively little change in the heart sound characteristics

Not all episodes of acute coronary artery closure are accompanied by the various aspects of the cardiogenic shock syndrome in some the pain factor may be mild and of short duration and few of the striking symptoms of the syndrome are evident There are also instances where the patient may be more or less unaware that significant changes have taken place in the cardiovascular system usually such myocardial infarctions are relatively small and are discovered accidentally on subsequent ECG examination or at autopsy It is not an uncommon experience in general post mortem practice to note the presence of extensive myocardial scarring due to long standing infarcted areas in subjects who have given no history of cardiovascular disability Routine ECG examination of all clinic and hospital patients over the age of 50 have likewise revealed a large number of abnormal tracings which have been interpreted to represent previous unknown or unrecognized coronary episodes It is probable that so called silent coronary occlusion and myocardial infarction is rather common The subject of such missed cases is currently under close investigation at several cardiac research centers

Acute coronary artery occlusion may also masquerade under a variety of clinical syndromes it has long been known that it may simulate the acute surgical abdomen with all of the leading symptoms of appendicitis gallbladder disease perforated gastric or duodenal ulcer and intestinal obstruction Renal stones on the left side however may simulate

the symptoms of coronary closure hiatus hernia may also produce similar findings In nearly all of the extra cardiac conditions a previous history of the specific disability can ordinarily be obtained or there may be definite evidence of the pathologic process responsible for the given attack

A further word about the hiatus hernia syndrome may be made in relation to diagnostic problems the condition is fully discussed in another chapter but in acute manifestations of this syndrome the onset of the pain pattern the shock nausea and vomiting may be very similar if not identical with that seen in acute coronary disease Moreover ECG changes occur in both conditions alterations of the ST segments and T waves are common during the acute phase of these disturbances

Of especial serious outlook are instances of dissecting aneurysms of the aorta particularly in the upper thoracic portion here the symptoms may mimic in every detail those of coronary closure and myocardial infarction occasionally the two entities may occur simultaneously The prognosis in the observed cases has always been fatal

Since the laboratory data associated with the development of heart muscle damage in acute coronary closure may require from 12 to 72 hours to reach significant diagnostic importance differential recognition of the various clinical conditions which may be confused with the syndrome must depend almost entirely upon the physician s skill and experience in the very acute phase of the disease In doubtful cases priority should of course be given to the most serious disability with antibiotic therapy there is less danger in extending the observation period in the acute surgical abdominal patients than formerly The outlook in acute myocardial

infarction during the initial shock stage is definitely not improved by laparotomy regardless of the minimal surgery involved Here the physician is faced with two serious pathological conditions the choice is between overwhelming infection and cardiovascular breakdown

TREATMENT OF ACUTE PHASE OF MYOCARDIAL INFARCTION

In a recent study of nearly 1000 cases of acute coronary artery occlusion and myocardial infarction made in New York City it was found that about 62% occurred outside of the patient s home The largest number developed during transportation from one place to another or directly after arriving at the destination the locale of the incident included subway cars buses taxicabs private automobiles railroad cars hotel lobbies department stores theatres and business offices Attacks while at various types of work usually developed some hours after arrival About 12% of the initial episodes occurred at night in bed while 18% of the secondary or subsequent closures also developed in bed at home or in hospitals

The question of removing the patient from the scene of the episode to a more suitable environment arises in most emergency cases Unless complete cardiovascular collapse has quickly developed transportation may be made by automobile with the patient walking or carried to the *front* seat of the vehicle the front seat is emphasized here since it has been shown by Karpovich and other physiologists that in most cars far less energy in manipulation is required to get into a front seat than in the rear ones Not all coronary cases require hospitalization but if a medical institution is closer

to the locale of the extradomiciliary attack removal to the hospital is preferable to the home

Appearance of shock and collapse demands on the spot medical care if cardiac arrest has already occurred the measures described in resuscitation of the stopped heart should be promptly applied Where pain is a predominating symptom one of the narcotic drugs should be administered intramuscularly there is no place here for the sedative or tranquilizer group The paroxysmal arrhythmias should receive appropriate management and if acute congestive failure appears with pulmonary edema specific therapy is indicated Unless cardiac arrest has occurred or is likely to take place the patient should be removed as expeditiously as possible by ambulance to a hospital Most ambulances are equipped for emergency oxygen therapy this may be administered *en route* to the receiving ward

There should be no delay in the admission procedures at the hospital it should be unnecessary to mention that certain mandatory regulations have in some instances been carried out at the expense of urgently needed time and the patient s welfare A 10 to 20 minute period of waiting for administrative compliance may spell the difference between life and death in critical conditions The importance of hospital admission rules and regulations is recognized but special provisions should be enforced concerning the availability of trained personnel ready to handle such cardiovascular emergencies with dispatch and without delay

CARDIOGENIC SHOCK

It has been estimated that about 82% of all myocardial infarctions due to coronary artery closure occur in the left ventricle and about two thirds of these are in the chamber wall With the loss of such important contracting areas ventricular output falls and forward failure develops The essential physiologic factor in lessened ventricular output is the fall in *functional* blood pressure in this context functional blood pressure is chiefly *pulse pressure* Experiments have shown that cellular metabolism of the vital tissues and organs of the body require a minimum of 15 to 20 mm Hg pulse pressure to maintain their normal physiological activity the brain heart kidney and endocrine glands are the most sensitive to lowered pulse pressure while the liver spleen lungs and skin are less responsive to subfunctional hypoxia The fall in functional pulse pressure is more important than the actual drop in blood pressure levels thus a fall from 160/90 to 90/68 mm Hg may appear to be more dramatic than a drop from 120/84 to 100/84 but in the former instance pulse pressure is 22 mm Hg and in the latter only 16 mm Hg which is within the cardiogenic shock levels

Pulse Pressure It may be difficult to determine pulse pressure when systolic levels fall to 40 to 60 mm Hg at this point only a single sound may be heard on auscultation with the conventional blood pressure apparatus It is not uncommon in such cases to note on the hospital record that the patient had *no* blood pressure This of course is not factually correct until cardiac arrest occurs ventricular contraction is accompanied by a measurable output Special equipment

may however be required to estimate such low pressure levels but simple palpation of the radial artery may reveal a systolic level when auscultatory methods fail If intra arterial injection treatment is contemplated the needle used for vessel puncture may be initially attached to a water manometer filled with isotonic solution the amplification here is about 13 to 1 compared to a mercury manometer Thus a clinically immeasurable blood pressure of 42/20 mm Hg becomes 520/260 mm H O small changes in ventricular output due to therapy may be readily estimated by this method

Minimal Ventricular Output During the period of minimal ventricular output the "vital organs previously mentioned demand attention and should receive as much of the available circulating blood volume as possible since the brain leads in priority full use of gravitational factors should be employed here In shock position the head should be lowered 12 to 16 inches below the level of the chest abdomen and legs If pulmonary edema is present this position may require some caution since the optimum position in orthopnea is in the reverse body plane with the head and chest elevated in relation to the rest of the body In balancing the orthostatic factors under such conditions the physician will have to exercise good judgment in determining the more important disability to be considered at the moment and in certain cases alternate priority must be given

The volume of available circulating blood may be controlled to some extent by restricting the circulation in the arms and legs this procedure has been described elsewhere but it may be repeated here Three conventional blood pres

sure instruments are desirable but tourniquets may also be employed The cuffs are inflated to a level about 10 mm Hg above the estimated systolic pressure application of the cuffs is made in the brachial and femoral areas of the limbs One of the limbs is free at any given time and hemostasis is maintained no longer than 5 minutes The cuffs are rotated around the 4 limbs at each inflation a period of 2 minutes is permitted between the compression intervals A simple test of the effectiveness of this method is the rise in blood pressure when the 3 limbs are deprived of their normal blood volume Unless there is a definitive increase in pulse pressure the procedure may be worthless as well as frustrating it requires the full time attention of trained personnel for satisfactory performance

Increase in pulse pressure may sometimes be secured by intra arterial injection a variety of injectable preparations have been used Here the vasotensor drugs are of special importance Norepinephrine or Levophed Bitartrate (Winthrop) Memphentermine Sulphate or Wyamine (Wyeth) Methoxamine Hydrochloride or Vasoxyl (B W & Co) and Phenylephrine or Neosynephrine (Winthrop) may be used together with glucose and saline buffered solution

Intravenous Therapy It is doubtful that intravenous injection serves any useful purpose at this stage of cardiogenic shock since there may be an actual hypervolemia at this time if pulmonary edema is present phlebotomy may be indicated The withdrawn blood may be re injected intra arterially if no oxygenating equipment is at hand it may be recalled that venous blood may still retain a relatively high level of available oxygen In the normal exchange mechanism

venous blood has as much as 14 to 16% oxygen saturation when ambient oxygen is about 20% under the conditions of increasing hypoxia in cardiogenic shock this may fall to 10% which is the critical level of functional tissue oxygen metabolism At this level such venous blood would require artificial oxygenation to be of clinical value

Oxygen Therapy It has previously been pointed out that the definitive treatment of hypoxia is the administration of pure (100%) oxygen the utilization of oxygen is dependent upon pulmonary function Pulmonary function in turn is based in great part upon the normal physiologic integrity of the exchange membrane mechanism of the lung alveoli In congestive failure with pulmonary edema membrane function is disturbed if the alveoli spaces are filled with fluid no or little gas exchange occurs The question has been raised by research workers in the field of space medicine and aerophysiology whether oxygen serves any useful therapeutic purpose when alveoli flooding occurs for any given reason clinicians have raised the same question in the pulmonary edema of cardiogenic shock

Pulmonary Edema Experience has shown that the empirical answer to this important problem is dependent upon the extent or degree of pulmonary functional involvement massive pulmonary edema with all lobes saturated is usually a terminal condition This however is far less common in myocardial infarction than in valvular heart disease like mitral stenosis The pulmonary edema in cardiogenic shock has a tendency to be more or less limited to the bases and lower lobes of the lung Vital capacity studies in this con

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Cardiovascular Investigation has applied the data discov ered by the Office of Climatology in the Weather Bureau in Washington to sickroom conditions and with special ref erence to cardiac patients The point of reference is in re gard to scientific patient comfort in terms of ambient tem perature and humidity The Hevener *Humiture Index* which is the numerical addition of temperature of the room and the actual humidity divided by 2 is readily available to the phy sician Thus if the room temperature is 80° and the humid ity is 50% the humiture index would be 65 which is within the comfort zone while a temperature of 75 and a humid ity of 90% would give an index of 82 5 which is far above the physiologic limit of patient comfort More complicated but more scientific is the recently proposed *Discomfort Index* or DI this determination requires a dry and wet bulb thermom eter apparatus and use of the formula

$$DI = 0\ 4\ (Td + Tw) + 15$$

thus when the dry bulb reading is added to the wet bulb reading and the result is multiplied by four tenths and 15 is added the Discomfort Index is obtained The upper normal limit of the index is 70 but in cardiogenic shock a better level is from 60 to 65

Oxygen Tents The use of oxygen tents presented a number of unnecessary problems which were of considerable disserv ice to the patient Much has been said about the psychogenic implications the chief of these is the development of claus trophobia which occurs in subjects isolated in a small com partment and practically shut out from the normal activities of life around them It is likely that many of the other neo

psychotic complications which harassed physicians nurses and the family in the previous treatment of acute coronary occlusion and myocardial infarction may have been precipitated or enhanced by close confinement in oxygen tents

Other phobias of varying importance have also developed in certain individuals the fear of removal from the tent is not uncommon Agonizing memory of the pain difficulty in simple breathing nausea and vomiting and a host of other disagreeable and alarming symptoms which accompanied the early hours and days of the heart attack may delay the patient s voluntary removal from the tent which has become a symbol of refuge and safety Like the use of the mechanical lung in bulbar poliomyelitis with respiratory paralysis patient dependency upon an artificial environment may pose a difficult problem in convalescence

Mask and Nasal Catheter In general oxygen is more efficiently administered by mask or nasal catheter ambient oxygen saturation in the usual tent service rarely exceeds 40% - it may reach 80 to 90% in adequate mask procedures and about 40 to 60% by catheter If the patient is irrational and thrashing around the catheter method is easier to employ than the mask there is however a certain degree of nasal irritation in prolonged use of the catheter Most conscious and cooperative individuals prefer the mask which may be self applied and removed at will The practical test of oxygen usefulness in any given case is the patient s willingness to continue its administration if the oxygen relieves the work of breathing lessens the intensity of the pain or somehow makes the individual *more comfortable,* without attempting to define the implications of being *more comfort*

able the procedure should be employed When on the other hand there is patient rejection for one reason or another oxygen will serve no useful purpose regardless of the academic concepts concerned with its employment

The objective evidence of oxygen benefits is obvious improvement in the hypoxial syndrome this may be indicated by lessening of orthopnea increase in pulse pressure decrease in the signs of congestive failure and the return of more normal general physiologic function The kidney in particular may serve as a guide or index of favorable response when pulse pressure reaches critically low levels renal function diminishes Urinary suppression is the rule in severe cardiogenic shock complete anuria may occur during the first 12 hours Thus any increase in urinary output suggests a more hopeful outlook

DISTURBANCES OF CARDIAC RHYTHM

Cardiogenic shock is usually accompanied by relative grades of sinus tachycardia with rates extending from 110 to 160 regular rates below 50 suggest complete heart block and regular rates above 180 may be due to atrial flutter or paroxysmal tachycardia Regular rhythms regardless of the rate are less likely to be as hazardous as the irregular types of cardiac cycle disturbance Simple sinus tachycardia ordinarily requires no special medication but the arrhythmias demand close attention The electrocardiograph is an essential tool in the management of acute coronary artery closure and myocardial infarction it is mandatory for the understanding of such irregularities as may occur It is of less importance in the determination of the specific area of the heart muscle involved Experience over the years has shown that

there is an unfortunate tendency among the readers of electrocardiograms both in hospital and private practice to spend much time and energy in attempted differential diagnosis of the various ST segmental and T wave changes which may occur in acute coronary artery disease since there may be no correlation between such findings and the clinical aspects of a given case and since there is no fundamental difference in treatment based upon localization such observations are chiefly academic Both anterior and posterior infarcts as well as apical lateral septal diaphragmatic and junctional involved areas demand the same therapeutic approach and the available statistical data does not indicate differences in complications objective symptomatology or mortality based upon localization

Identification of Disturbances of Rhythm When these occur it is however of considerable clinical importance here there is a relatively wide choice of drugs and other measures *which may be specific in their effectiveness in controlling* certain undesirable and perhaps lethal complications caused by certain of the irregularities developing during the cardiogenic shock phase of acute myocardial infarction

Premature Beats The most common disturbance of rhythm are premature beats supraventricular extrasystoles are far less significant than those arising from ectopic foci in the ventricular areas There is no correlation between the frequency of the premature beats and the eventual outcome in any given case the inherent danger in every ectopic focus is *the factor of its rhythm domination potentialities* A single beat may have the same potentiality to initiate a *dominant*

extrasystolic rhythm as more frequent ectopic beats There has been a decided change in the concept of premature beat significance in acute myocardial infarction in the past decade older authors considered ectopic beats from the point of view of frequency on the postulate that more premature beats were of greater significance than isolated or few such extrasystoles Such an observation may be misleading all premature beats arising during acute myocardial infarction must be regarded with some suspicion and measures should be undertaken to suppress their development Many such ectopic beats fortunately remain relatively innocuous but some may lead to functional cardiac arrest and death

TREATMENT OF PAROXYSMAL TACHYCARDIAS

Management of the paroxysmal tachycardias which may develop from ectopic foci arising in or near infarcted areas of the heart muscle as well as the other common disturbances of rhythm like atrial flutter and fibrillation heart block bundle branch dysfunction and ventricular flutter and fibrillation is no different than when these occur under other cardiovascular conditions Detailed discussion of these arrhythmias and their treatment are presented elsewhere in this book

Treatment should be started promptly after diagnosis and continued vigorously until favorable results have been secured It should be pointed out here that during the cardiogenic shock period all medication is *parenteral* since gastrointestinal function is at a standstill or reduced to subfunctional levels it is doubtful that there is any or little drug absorption when given by mouth Dosage under the circumstances can not be calculated with the accuracy demanded

at this critical stage of the syndrome The intravenous route is first choice when congestive failure is present hypodermic and intravenous absorption may be delayed or potentiated unfavorably by extracellular accumulations of fluid No injection of course should ever be attempted in obvious areas of edema this applies particularly to the thighs and buttocks in anasarca

ACUTE CONGESTIVE FAILURE

In cardiogenic shock congestive failure may develop rapidly after the initial few hours of the syndrome pulmonary edema is the most common finding Starting with a few crackling rales scattered throughout the lungs fluid may accumulate quickly in certain instances both lungs may become involved within 10 to 30 minutes A constant frothy cough sometimes more or less blood streaked increases the dyspnoeic factors and renders the outlook more guarded This constitutes one of the most critical phases of the acute myocardial infarction syndrome the mortality here is high The pathology is basically due to more or less complete left ventricular failure, Brooks spoke of the heart off balance under such circumstances The right ventricle continues to function with a developing increase in pulmonary pressure unless pulmonary hypervolemia is reduced by left ventricular output fluid tends to accumulate in the alveolar system of the lungs

EMERGENCY TREATMENT

This must be directed to both sides of the heart the left must be supported by increasing the functional contracting effort of the failing heart muscle and the right by reducing venous back pressure In patients where pulmonary edema

has reached dangerous levels phlebotomy should be per formed first about 300 to 500 cc should be removed quickly If possible this blood should be handled with sterile pre cautions it may be used later for intra arterial injection While the blood is being withdrawn intravenous injection of one of the digitalis or strophanthin preparations should be administered in the other arm Full calculated dosage should be given there is no place for timidity here Aca demic speculation in regard to the possible effect of in creased tonus and muscle contraction of the myocardium involved in the infarction process will only delay the pos sibility of a favorable therapeutic response The calculated risk is in relation to possible rupture of the heart or the de velopment of emboli Such instances have been reported but it has not been seen in the author s experience on the other hand a number of instances have been seen where a fatal outcome has occurred when digitalis has been delayed or omitted because of such theoretical considerations Stro phanthin in the hands of those familiar with its mode of action may be life saving in overwhelming pulmonary edema of this type the drug has received far less attention in this country than in Europe The margin of safety is small and the difference between full therapeutic effect and over dosage requires considerable experience in its use

The place of intravenous (aminophylline) in cardiogenic shock types of acute pulmonary edema is questionable no harm apparently results from its administration Beneficial effects are more likely to occur if the patient has had previ ous pulmonary or bronchial disability In concentrated dos age it may have a selective vasodilator action on the coronary artery tree and insofar as any improvement in heart muscle

circulation will enhance recovery it may have an area of usefulness

ANTICOAGULANT TREATMENT

No drugs in modern medical history have excited such controversial discussion as the anticoagulant preparations after more than 12 years of clinical trial the two schools of thought are still in violent disagreement This is unfortunate for the same collected statistics are used by both groups to prove their standpoint The physician would be wise to choose a wide middle course in which *not all* patients with acute coronary occlusion and myocardial infarction are given anticoagulants nor *all given none* The indications for such therapy have become more definitive as the years have rolled by and as clinical experience has accumulated

Indications From accepted statistical data there can be no question but that anticoagulants lessen the frequency of embolization when disturbances of rhythm occur in cardiogenic shock there is also no question that when embolization has occurred even with sinus rhythm the incidence of secondary embolization is materially reduced Finally if the hypotensive factors dominate the clinical picture the drugs may prevent peripheral arterial thrombosis On the other hand if cardiogenic shock is not present and the patient's symptomatology is minimal with no objective evidence of cardiac arrhythmia it is doubtful that anticoagulant therapy is indicated if the patient under these circumstances has had previous episodes however prudence suggests the employment of the drugs In the symptom free individual who has experienced a fleeting attack of pain followed by confirma

tory ECGs and laboratory data anticoagulant treatment may have no place

It may be said at this point that there is no problem presented in starting anticoagulant therapy if it is indicated the major problem occurs in determining *when* to stop Here a host of minor and major considerations will plague the physician this phase of anticoagulant therapy is discussed elsewhere in this volume but it must be borne in mind in the doubtful and borderline case

Heparin Anticoagulant therapy should start with the use of the heparin series of preparations given intramuscularly in cardiogenic shock it may be administered intravenously In the latter condition and in the absence of gastrointestinal function parenteral injection must be continued until the patient is able to take food by mouth in severe cases this may take as long as 3 to 8 days The hydroxycoumarin drugs may then be given in sufficient dosage to maintain the optimum prothrombin time ratio

The chief disadvantages in continued anticoagulant therapy are two technical and psychogenic The technical problems are concerned with constant prothrombin control measures in hospitals and larger communities where laboratory facilities are available the question of expense and personal inconvenience insofar as the patient is concerned may have important implications On the other hand where regular laboratory check up is impossible the drugs are given with some hazard of overdosage and hemorrhage Likewise too little dosage serves no useful purpose and may confuse subsequent understanding of the clinical picture one instance out of many may be illustrative A traveling sales

manager age 56 experienced a severe coronary episode which required a long convalescence before he was able to resume his previous work he remained under satisfactory prothrombin control during this period but the difficulties of obtaining laboratory service in the various cities and towns listed on his traveling schedule soon broke the rigid routine which had been maintained He continued with his usual dosage of Dicumarol however for several weeks when he had another attack in a city far from home on admission to a local hospital the episode was confused with an acute pleuritis on the left side In view of the history of continued anticoagulant therapy, the physicians directed more attention to the pleuritic symptomatology until it was found that his prothrombin time was within the normal control of 14 seconds Undue confidence placed upon the story of anticoagulant therapy delayed recognition of the new attack for almost 2 days

The Psychogenic Problems Equally as disturbing as the technical are the psychogenic problems here both the physician and patient are involved for as indicated above the question of when if ever to stop the drugs can not be answered easily or satisfactorily There is a great burden of clinical responsibility placed upon the physician in stopping the anticoagulants after any given period of administration be it short or long if he decides that a patient who has been symptom free from his previous coronary episode for months or years is now ready for discontinuance of such therapy and it is stopped accordingly and shortly thereafter the individual experiences another episode or develops an emboliza

tion syndrome presumed cause and effect are likely to be emphasized by the patient and his family The threat of subsequent cardiovascular disability is unfortunately inherent and implied in any attempt to stop such anticoagulant preventive therapy and great moral as well as clinical courage may be required in the consideration of any given case

The indications for continued anticoagulant therapy are likewise two repeated episodes of myocardial infarction and repeated evidence of embolization If the patient has signs of advanced atheromatous disease with generalized vascular pathology involving not only the coronaries but also the cerebral renal and peripheral vessels and particularly if hypertension is present such therapy may have to be continued indefinitely Life expectancy under such conditions is limited perhaps to 3 to 5 years regardless of the age period of the initial myocardial infarction Individuals who develop paroxysmal tachycardia or intermittent atrial fibrillation following an attack are definite candidates for long continued anticoagulant measures 4 of the author s original series of 12 cases of severe cardiogenic shock reported in 1953 are still alive after 5 years of anticoagulant treatment but with minimal functional capacity

Anticoagulant Drugs Finally a word about the choice of the anticoagulant drugs In the heparin series the following have been used successfully in hospital and private practice Heparin Sodium (Lilly) Liquaemin Sodium (Organon) Depo Heparin Sodium (Upjohn) Heparin Repository (Philadelphia) and Hepo Nine B Fortis (Columbus) the latter contains heparin sodium choline chloride and Vitamin B 12

a combination which has a clearing action in relation to the giant molecules of cholesterol seen in certain types of atheromatous coronary artery disease

The list of hydroxycoumarin drugs continues to increase with pharmacologic research enterprise but each may have an appropriate place in the treatment of any given case The best known of these are bishydroxycoumarin or Dicumarol (Abbott) phenidione or Hedulinx (Walker) ethyl biscoum acetate or Tromexan (Geigy) diphenadione or Dipaxin (Upjohn) phenprocoumon or Marcumar (Hoffman La Roche) acenocoumarin or Sintrom (Geigy) and cyclocumarol or Cumopyrin (Smith) *New derivatives of warfarin are* Coumadin (Endo) and Prothromadin (Harvey)

Overdosage in anticoagulant therapy is indicated by marked increase in prothrombin time estimations in the authors experience the optimum *clinical levels should run* about twice normal *or from 26 to 36 seconds* This may be noted on the patients chart by the Alsahy formula in which the determined prothrombin time is placed over the normal control as in writing a fraction thus if the prothrombin time on therapy is 28 and the control 14, this may be charted as 28/14 Higher levels than the 2 to 1 ratio may be necessary in certain individuals but rarely over an Alsahy 45/14 levels over 50/14 may become hazardous in relation to possible capillary hemorrhage Purpuric spots on the skin subconjunctival hemorrhages and hematuria are clinical signs of overdosage

Prothrombin function can fortunately be restored to within normal limits more or less rapidly by the Vitamin K preparations both natural and synthetic substances are usu

allv effective except in extensive cerebral bleeding Adrestat Mephyton and Synkavite are all equally recommended

GASTROINTESTINAL TRACT IN CARDIOGENIC SHOCK

One of the most disturbing complications in cardiogenic shock following acute myocardial infarction is the nausea and vomiting which may develop early in the attack indeed an overwhelming nausea may actually precede the onset of the pain factors It was this type of nausea which predominated the clinical picture of so called acute indigestion familiar to the older physician before the syndrome of acute coronary closure had been clearly defined about 40 years ago in the literature of the 1900 s acute indigestion was described as a serious and oft times fatal condition

Nausea and vomiting mav also be initiated by the administration of morphine given for the relief of heart pain in responsive subjects a dose as small as 1/4 grain may cause nausea In others repeated doses may have been given because of the intense pain factors control of such pain may be at the expense of stimulating the vomiting center of the brain Many patients in retrospect have complained more of the nausea and vomiting which occurred than of any other symptom pain and dyspnea are quickly forgotten but memory of the retching and nausea may be retained for some time For this reason as explained earlier morphine is the least desirable of the narcotic drugs to employ Demerol Dilaudid Pantopon and diethyl morphine should be selected

The nausea and vomiting is ordinarily a manifestation of the hypoxial syndrome both at the central nervous system level and local at the cardiogastric plexus it occurs in experimental animals during cardiac standstill and may have a certain *teleologic* significance Vomiting is chiefly a reflex vagal mechanism involving violent contractions of the stomach esophagus diaphragm and upper abdominal muscles such contractions may have physiologic implications in regard to *coronary blood flow* In cardiogenic shock all gastrointestinal activity ceases below the sphincter and abdominal distention may follow

Elevation of the diaphragm in *paralytic ileus* regardless of the cause poses a special threat in myocardial infarction reduction in lung space may seriously reduce vital capacity function Where orthopnea is already present limiting pulmonary ventilation may do much to increase the tendency for the *development of pulmonary edema* As manifestations of reflex disturbances of the gastrointestinal tract both vomiting and visceral distention may thus seriously aggravate the already overburdened hemodynamic system and render treatment more difficult

It is advisable therefore in cardiogenic shock to permit nothing to be taken by mouth there is no objection to occasional rinsing of the mouth if the patient can cooperate With alimentary digestion at a standstill even water may not be absorbed and the temptation to give frequent small drinks may do more eventual harm than good The Viennese school of cardiologists were perhaps the first to exploit the *starvation theory* in such cases with certain *modifications it* should be routine where nausea and vomiting both with and

without abdominal distention are predominant complications

LABORATORY DATA

Positive Tests The humoral or general constitutional physiologic response to acute coronary artery occlusion with myocardial infarction can not ordinarily be determined until several hours after the onset of the episode some changes may require several days before definitive information can be obtained However in most cases confirmatory laboratory evidence of the syndrome is possible within the first 24 to 48 hours in the battery of tests available in most hospitals several types of examination may become *positive* within this period Positive is used in this context to mean tests which are changed from normal and in the absence of any other etiologic factor to explain such alteration are interpreted to be the result of or caused by acute myocardial infarction The definition is spelled out in detail to emphasize that all laboratory data must be considered within the framework of the lesion under study if other pathologic conditions are present and in any way contribute to or detract from the given laboratory results the test is no longer valid or definitive insofar as the cardiac lesion is concerned

Very early positive tests must thus be regarded with some suspicion in most instances such data suggests a pre existing condition probably extracardiac in origin A high white blood cell count or a high sedimentation rate discovered during the first 8 to 12 hours is not likely to be due to the myocardial infarct Marked ECG changes in the early hours of an attack suggest previous heart disease while elevated transaminase levels point to other causes for this finding

Prompt Laboratory Studies However much is to be said for the prompt determination of all of the laboratory examinations since it is well recognized that significant changes do not develop quickly in myocardial infarction there is a natural tendency among some clinicians to delay these tests until they are more likely to be positive Experience has shown that much information concerning the *status quo ante* of the case may be lost by this concept any data which can be obtained in regard to the patient prior to the present attack may place subsequent tests in a more useful perspective For example in the illustration given above concerning the discovery of ECG abnormalities early in the episode subsequent changes if they occur may be more accurately evaluated in terms of the prior findings Such *status quo ante* factual data is especially important when a given patient has experienced a previous coronary attack unless known and recognized such residual changes may add confusion in the interpretation of the new tracings

Correlation Factors There is an unfortunate lack of correlation between laboratory data and the actual extent and severity of any myocardial infarction marked changes from normal in the various tests do not necessarily indicate that the lesion is a large one The reverse is equally true small or minor changes do not rule out extensive myocardial damage A high leukocytosis for example of 22 000 WBC has no more clinical significance than 12 000 or 15 000 likewise a sedimentation rate of 60 is no more informative than 30 or 40 The former Cobb rule of thumb in which the gravity of the syndrome was estimated by the degree of humoral re-

action has no validity in the light of increased knowledge of the pathologic physiology involved in the mechanism of arterial occlusion and muscle infarction Cardiogenic shock and death have occurred in patients with more or less normal white cell counts sedimentation rates ECG tracings and transaminase levels

This should not be taken to suggest that laboratory data is unimportant it means that the physician must use judgment and experience in the *clinical* interpretation of all laboratory procedures Assuming that no technical errors are suspected two questions must be asked in every case first is there any other condition which the patient has now or has had in the past which could produce the given results and secondly do the results confirm the clinical impression or diagnosis? These questions might well be asked in regard to any acute disease process but in myocardial infarction they may be of special importance in differential diagnosis

ELECTROCARDIOGRAPHIC CHANGES

It has already been said that the electrocardiograph is one of the important tools in the examination of patients with myocardial infarction it has also been pointed out that the ECG tracing *per se* may neither be diagnostic nor prognostic insofar as the syndrome is concerned The electrodynamic events of the cardiac cycle may have little or no correlation with the clinical aspects of the individual However any change in the previous pattern of the ECG may have certain implications which may be evaluated at the same level as any other pertinent laboratory test

Mechanism of ECG Changes Assuming that a given subject has had a more or less normal ECG pattern before the attack electrodynamic alterations are most likely to occur in that part of the cycle concerned with the repolarization process It can be shown that ischemic changes in the musculature of the left ventricle may produce a disturbance in electrolyte metabolism so that the normal or usual summation of cellular potential is diminished or lost The normal ST segment at its neutral or iso electric level and the T wave with its positive or upward deflection may be altered by such electrolytic changes When myocardial infarction occurs the electric potential differences developed by the involved areas may neutralize or dampen the electrodynamic gradient factors in the repolarization of the remainder or non injured part of the heart muscle or it may entirely dominate this phase of the cycle If the damaged area is located in those parts of the myocardium which have certain physiologic isolation factors no involvement of the repolarization process may occur

While this is a greatly oversimplified explanation of the ST segmental and T wave changes seen in myocardial infarction and is subject to a number of academic corrections it may serve as a practical background for the clinical use of the ECG tracing It emphasizes the important point that there may be no correlation between actual size of the infarcted area and the extent or degree of changes seen in the ECG record it also serves to explain why myocardial infarction may also occur without demonstrable ECG alterations

The ST segmental and T wave changes which are seen in acute myocardial infarction ordinarily require from 8 to 48 hours to develop some times full development of the abnor

mal pattern may take several days This is interesting in view of the immediate changes which occur in the Master exercise tolerance test in responsive subjects similar ST segmental and T wave changes may occur directly after physical effort (climbing stairs) Restoration to the pre exercise pattern may require from 3 to 10 minutes It has been postulated that since the electrodynamic changes are the same the basic physiologic mechanism responsible for the disturbance of repolarization must be more or less identical Whereas the alterations after exercise represent a temporary and reversible process myocardial infarction is permanent and irreversible This does not answer the question however concerning the delayed electrodynamic response in heart muscle damage except to point out the difference between *total* myocardial ischemia and *localized* muscle involvement This is more than academic discussion the practical implications have a clinical application which must be considered in the interpretation of post infarction ECG tracings

Benign T wave Changes There are many benign causes of T wave abnormalities in the author s paper Clinical Implications of Electrocardiographic Misinterpretation Some Observations on T Wave Abnormalities (New York State Journal of Medicine 57 2659 1957) it was shown that the simple drinking of iced water as well as post prandial effects may produce inverted T waves there are many psychogenic conditions which cause similar changes The fright electrocardiogram" is a well known phenomenon and pain itself may be responsible for T wave abnormalities There are thus many pitfalls in the interpretation of the repolarization pattern and a careful evaluation of all the factors concerned in

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these ECG changes and prognosis in any given case renders the discovery of such alterations to the level of any other laboratory report It may be emphasized at the expense of frequent repetition that patients die in cardiogenic shock with no changes in the ECG tracings while others make a rapid recovery with grossly abnormal ECG patterns

ROLE OF ELECTROCARDIOGRAPHY

Most cardiologists agree that the most important role of the electrocardiograph is the identification of the disturbances of cardiac rhythm which may develop in acute myocardial infarction while an experienced examiner may make a pre sumptive diagnosis in most instances the definitive mech anism responsible for the irregularity may require instrumen tal analysis This is particularly true in the paroxysmal tachycardias drug treatment clinical management and prognosis may be entirely dependent upon the differential diagnosis between paroxysmal atrial flutter and fibrillation and the rhythms due to nodal or ventricular tachycardia It may be impossible to distinguish clinically between frequent premature beats and atrial fibrillation or between a sinus bradycardia in the range of 40 to 48 beats per minute and complete heart block A sinus tachycardia in the 150 to 160 range may be mistaken for an atrial flutter with a 2 to 1 block Ectopic beats which occur in atrial fibrillation may be missed finally it has been previously pointed out that skipped beats of incomplete heart block are easily confused with simple extrasystoles For the identification of all of these the ECG examination is of course necessary and no effort should be spared to determine the exact type of ir regularity

Number of ECG Leads How many leads should be taken in the examination and study of acute myocardial infarction? The question is of more than scientific and academic interest in any given case there are the implications of time expediency disturbance of a very ill patient and perhaps technical problems In the irregularities of rhythm and in the conduction abnormalities one lead may suffice experience has shown that either Lead 1 or 2 alone may be employed satisfactorily In a subject who is thrashing around in pain or with uncontrollable nausea and vomiting it may be impossible or undesirable to obtain more than one limb lead either conventional or unipolar Precordial leads may be difficult to secure in restless orthopneic and otherwise uncooperative individuals If however the conditions are such that the physician is able to make a complete ECG examination without too great manipulation or unusual difficulty the opportunity should be seized to take the standard 12 lead hook up this would include the 3 conventional limb leads the 3 unipolar limb leads and the 6 V leads In certain instances parallel CF leads should also be taken if the major lesion is predominantly in the anterior vector planes In special cases where posterior infarction is suspected and where the anticipated changes in Leads 2 3 aVL aVF and posterior V 8 and 9 or CF 7 and 8 are debatable esophageal leads may be diagnostic and should be attempted provided the procedure meets with patient acceptance and cooperation There is thus no specific answer to the number of leads that should or must be taken the practitioner will be guided chiefly by the information secured in the available leads He should however continue to explore until he has

the data necessary for understanding and managing the situation

ASYMPTOMATIC MYOCARDIAL INFARCTION

It has already been stated that the scars of old myocardial infarcts is not an uncommon discovery at post mortem examination of patients who have died from extra cardiovascular causes and in whom no history of previous heart disease has been obtained How many such *silent* infarctions are experienced by the general population is difficult to estimate but competent pathologists have reported that in subjects over the age of 60 the incidence may run as high as 48% It is possible therefore for an individual to experience an episode of acute coronary occlusion with myocardial infarction without symptoms severe enough to call for medical attention these cases may be picked up subsequently on routine ECG examination or by the accidental discovery of aneurysmal changes of the left ventricle seen in x ray films of the chest

Minimal Symptoms Between this group and those who promptly develop cardiogenic shock there is a large number of individuals who suffer myocardial infarction with a minimum of symptoms or complaints A slight episode of precordial or substernal pain or pressure perhaps some dyspnea with sweating followed by a general feeling of weakness for a few hours or perhaps a day or two may complete the entire clinical picture If the patient has had previous attacks of angina pectoris this particular episode may be considered

as just another occasion for the use of nitroglycerine or his habitual remedy in others this initial or new experience may lead them to call the physician for medical assistance Since the acute phase of the attack may be of short duration the physician may find little in the physical examination However a drop in blood pressure when former levels were normal or elevated a tachycardia of 110 and above poor and distant heart sounds compared to a previous examination the appearance of a new systolic murmur or changes in an old murmur the development of an arrhythmia or the discovery of an equivocable pericardial friction rub may all be objective findings suggesting diagnostic possibilities which the alert examiner will utilize Such individuals warrant observation for at least 3 or 4 days with house confinement but not necessarily bed rest during this period changes in the physical examination may be noted and appropriate laboratory studies should be made with particular attention directed toward relative rather than absolute changes which occur

Disappearance of Symptoms More difficult to manage are those patients who experience more or less severe symptoms for a few hours and then become entirely symptom free insofar as their subjective complaints are concerned Here there may be considerable objective evidence of the syndrome with unmistakable clinical and laboratory confirmation of myocardial infarction Active men of the so called rugged and vigorous type usually chafe at the restrictions placed upon their bed confinement and they may defeat the very purpose of bedrest by uncontrollable mental and physical agitation It may require the limit of the physician s persua

sion and professional efforts to handle such a situation Bessers dictum of justice tempered with mercy may serve the physician well under such trying circumstances when the patients effort in walking to a nearby bathroom is accomplished with less mental and physical expenditure of energy than the use of a bedpan and when sitting in a chair involves less provocative implications than assumed bedrest certain compromises with conventional and accepted treatment may be permitted

Psychogenic Factors Practical experience has shown that the outlook in such cases is not materially jeopardized by the activity granted by demands of the situation On the other hand peace of mind and mental adjustment to the problems involved in the recovery period of myocardial infarction has in some instances hastened the *subacute* period Subacute period is here defined as the interval from admission to the hospital or confinement at home until the usual 6 weeks have elapsed In this connection it is difficult to define the *emergency period* in myocardial infarction in the group under discussion complications rarely occur after 4 to 5 weeks On the other hand if the patient has experienced a severe episode of cardiogenic shock such an emergency status may be present for 90 days or longer

ADDITIONAL COMMENT

Diet While the acute manifestations of acute coronary artery occlusion and myocardial infarction have been discussed in detail sufficient to cover the broad aspects of the syndrome space does not permit consideration of many minor but none the less disturbing problems which may arise in

the management of any given case The important question of diet has hardly been touched upon but this may be raised to a level out of proportion to its actual significance both by the patient and his family Salt free and salt restricted diets are indicated in congestive failure but it is doubtful that they serve any useful purpose in any other complication of the syndrome Adherence to such diets requires complete patient cooperation and simple recognition of this observation must be understood by every one associated with the care of the individual cheating and evasion of the basic electrolyte principles involved are not uncommon in tasteless and monotonous diets and every attempt should be made to make the patient s food tray as attractive as possible There are any number of practical and useful small books concerned with salt free diets these may be read with profit by those members of the family responsible for this service The question of cholesterol low diets comes within the same category in patients with high blood cholesterol levels and when there is objective evidence of atheromatous disease, such diets are important and should be strictly enforced For others, restriction of the saturated fat foodstuffs may be significant in overweight subjects as well as patients with diabetes In diabetic patients the usual dietary routine should be maintained

IV PREMATURE BEATS AND EXTRASYSTOLIC ARRHYTHMIAS

The most common *abnormal* disturbance of cardiac rhythm is due to premature beats or extrasystoles abnormal is italicized to emphasize that the most frequent of all types of irregular rhythm is sinus arrhythmia which is a *normal* finding in children and young adults Sinus arrhythmia is a vagal phenomenon associated with the respiratory cycle an increase in rate occurs on inspiration and a slowing down of the cardiac rate follows the expiratory phase In certain subjects there may be such an abrupt change in rate that a marked irregularity is noted Unless carefully observed such an irregularity may be misinterpreted clinically as a premature beat

Sinus arrhythmia is more common during slow rates it may entirely disappear when simple sinus tachycardia in the range of 110 to 140 occurs After exercise for example it is rarely seen it is lost also during hyperpyrexia regardless of the cause It is thus not usually seen during the fever stage of the acute infections but it may persist in acute rheumatic fever even with cardiac rates of 120 to 130 Sinus arrhythmia is ordinarily lost in the late teenage period but occasionally it may be discovered in the 30s and rarely in the 40s It is generally identified in relation to its association with the

respiratory cycle the subject is instructed to take a deep breath and hold it Increase in rate is readily observed In *some instances an ECG examination may be required for* definitive diagnosis it may be confused with premature beats as noted above

Premature beats may arise from any ectopic focus within the atrial or ventricular heart muscle they may also develop in or near the pacemaker areas A rare origin may occur in the septal tissues Epicardial and endocardial foci become operative by continuity with adjacent heart muscle tissue Ectopic beats may occur infrequently sometimes only 1 to 10 in 24 hours more often they develop several times a minute *When they follow each normal cardiac cycle coupled rhythm* is noted this may be an important finding in digitalis overdosage and myocardial infarction Premature beats may occur in a series and dominate the rhythm of the heart the mechanism of the paroxysmal tachycardias may be due to such continuous ectopic beats

CLASSIFICATION OF PREMATURE BEATS

Three Types of Classification Premature beats are conventionally classified in accordance with their anatomic point of origin sinus nodal atrial A V nodal ventricular and septal ectopic beats can be identified by ECG examination Premature beats may also be classified on a functional basis which is dependent upon their prematurity in relation to the diastolic interval of the cardiac cycle *Finally they may be* classified in relation to their hypoxial effect in coronary insufficiency Each of these classifications requires a word of clinical explanation

Site of Ectopic Focus The specific point of origin has significance in regard to practical evaluation and possible treatment as a rule the supraventricular ectopic rhythms are of far less clinical importance than atrioventricular nodal and ventricular extrasystoles Sinus nodal and atrial premature beats usually follow the same conduction pathways as do normal sinus released stimuli since they pass through the junctional mechanism of the conduction system they meet with the same blocking phenomena which is exhibited in atrial flutter and fibrillation They will also be controlled by the action of the digitalis drugs in this area On the other hand ectopic foci which arise below this area and this includes all of the A V nodal and ventricular premature beats are more difficult to manage since they are the product of potentially greater irritability factors of or within the myocardium Here the quinidine and procaine amide hydrochloride drugs are employed for their suppressive action The anatomic classification thus offers indications for selective therapy

Functional Mechanism The functional classification is important in the evaluation of the uneconomic physiologic factors produced by extrasystoles As noted before all premature beats occur during the diastolic filling period of the ventricles unless a sufficient volume of blood is within the ventricular chamber at the time of contraction the aortic valves will not open and no blood leaves the heart The pressure gradient within the left ventricle must exceed that of residual diastolic pressure above the aortic valves by at least 8 mm Hg before any volume output is hydraulically possi

ble The volume of blood in the ventricle at the time of ectopic contraction is thus a function of prematurity It should be pointed out here that multidiscipline graphic records are required to demonstrate the hemodynamic and electrodynamic relationships in many disturbances of rhythm, the ECG tracing alone does not reveal changes in the hemodynamic cycle The arteriogram or pulse tracing must be taken synchronously with the electrodynamic cardiac cycle However simple clinical examination may frequently disclose the functional response of certain irregularities in premature beats for example auscultation of the heart and palpation of the radial artery will indicate whether the ectopic beat has been effective and functional or whether it has been ineffective and thus a useless ventricular contraction The difference between the apical rate and the rate at the radial artery is known as a *pulse deficit* Pulse deficits are also seen in decompensated atrial fibrillation and flutter and other disturbances of rhythm Experiments have shown that when pulse deficit exceeds 10 beats per minute in any irregularity of rhythm an uneconomic functional output of the ventricle has developed it means that the heart has made a number of useless contractions from which no systemic benefit has been derived This number may be very large in a given 24 hour period with 10 such non functioning beats per minute there will be 1440 If the marginal reserve has already been depleted by hypoxia in any cardiac disability the loss of these ineffective beats may assume clinical significance

Hypoxial Effects Finally premature beats may be classified in relation to their hypoxial effects when there is considerable loss of coronary function This is purely an ECG finding the

T wave of the next normal sinus beat following the ectopic contraction may show changes which extend from a simple flattening or drop in amplitude to complete inversion In most instances the T wave will have recovered its original pattern in the second normal beat but in certain cases even the second and third cardiac cycles may show the effects of the premature beat It should be said that the interpretation of this finding is still open to further study some authors believe that other factors may be responsible for such T wave alterations but since the condition is seen almost exclusively in coronary insufficiency and in some types of myocardial toxicity like diphtheria acute rheumatic fever and hyperthyroidism it remains a significant finding in the extrasystolic arrhythmias

SYMPTOMATOLOGY OF PREMATURE BEATS

The clinical reaction to ectopic beats varies from patient to patient an entire gamut of subjective complaints may be described Some individuals are totally unaware of their occurrence even when brought to their attention they have no reactions or subjective symptoms regardless of the frequency of the irregularity Others may be greatly disturbed and terrified premature beats may produce a peculiar sensation in the epigastric region far removed from the heart and confused with gastrointestinal conditions Some patients describe a disagreeable throbbing in the head a pounding in the ears a fullness in the eyeballs a tug in the throat or a tightness in the chest Only occasionally is the ectopic beat associated with painful cardiac symptoms but in certain responsive subjects an ominous sense of palpitation or knocking of the heart occurs

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by premature beats Patients frequently volunteer the information that they always experience palpitation or skipped beats after eating this or that item of food such notations as "onions always give me heart burn and palpitation I can not drink coffee because I get extrasystoles I give up smoking because of heart beats and ice cold drinks give me palpitation are common on the history cards in cardiac clinics

Psychogenic Stress This too is a common predisposing cause of premature beats sorrow anger and excessive jubilation may be accompanied by extrasystoles The sudden death of his young nephew brought a physician in recently with a marked extrasystolic arrhythmia a contestant on one of the current give away television programs won a mink coat and was carried to a nearby hospital with an irregularity which was first diagnosed as paroxysmal atrial fibrillation She had frequent premature beats which persisted for almost a week

Physical Strain Occasionally excessive or unusual physical strain may precipitate ectop c beats in normal subjects a business executive was forced to change a tire on his automobile He was alone at the time and the effort required to jack up the car and other manipulations produced palpitations which demanded emergency care A healthy young mother was teaching her little daughter to jump rope but was forced to quit because of a pounding in her chest which was found to be due to ventricular premature beats

Extrasystoles also occur at night in normal individuals while they are in bed some patients complain that they tend

to occur when lying on the left side Others state that they *can not take food or drink before retiring, the exact opposite* is reported by those in whom an empty stomach always has a tendency to produce extrasystoles at night

Such tendencies, either real or fancied offer important clues in the management of symptomatic premature beats when they occur in otherwise normal individuals and the physician will exercise good judgment in careful appraisal of all pertinent facts concerned in a given patient s history Exclusion of the offending dietary substances and readjust ment of the subject s mode of living working and relaxation may be sufficient to control the symptomatology

Definitive therapeutic approach in the suppression of pre mature beats is unfortunately neither specific nor satisfac tory Where there is a large element of psychogenic factors treatment with the tranquilizer group of drugs may be re warding here in selected cases, self medication may be *advisable The patient is instructed to take the medication* only if and when he *feels* that he requires it Recent experi mental studies in this group have revealed a number of in teresting facts the first is that some types of symptomatic ectopic beats are more or less completely suppressed The second is related to diminishing the subjective reactions to the extrasystoles even though they continue to be present in some frequency the patient is unaware of their occurrence This has considerable clinical importance in the familiar well read or self taught group who develop the habit of constantly feeling their pulse in this difficult group to manage come doctors nurses dentists and other more or less medically trained personnel Pulse taking by any patient sooner or *later becomes* a vicious habit misinterpretation exagger

ated emphasis and unnecessary alarm is the rule and the physician should devote all of his energies to discourage this tendency except in certain isolated instances later to be described

Tranquilizer Drugs The former widespread use of the barbiturate drugs has decreased with the introduction of the tranquilizer preparations the slowing of mental acuity and the hangover effect in many patients may outweigh the beneficial results anticipated in either suppression or reducing the awareness of the irregularity It may however be necessary in a certain small group of individuals to employ the various preparations of the barbiturates when the meprobamate series have failed after a sufficient test of their effectiveness Alternate use of both types of medication has proven successful in some individuals

Digitalis and Strophanthin These should not be used in the control of simple premature beats regardless of the subjective symptomatology Quinidine and procaine amide hydrochloride should ordinarily be restricted to the management of extrasystoles which develop during acute coronary occlusion and myocardial infarction

Other Drugs A number of potent drugs have been suggested for the suppression of the extrasystolic arrhythmias which do not respond to the measures outlined above in certain selected cases under careful supervision isoproterenol hydrochloride or Isuprel (Winthrop) has been used successfully Methoxamine hydrochloride or Vasoxyl (B W & Co) has also been employed Both of these substances may produce toxic reactions even in small doses

In summary it may be said that while premature beats are the most common type of cardiac complaints their over all significance except in acute myocardial infarction is chiefly in relation to patient responsiveness to the irregularity The protean manifestations of the syndrome lends itself to management by many methods each of which may be tried in turn until eventual relief by one mechanism or another is achieved No two patients will respond to the same measures and the skill and ingenuity of the physician will be taxed in securing the desired results in some cases

V ATRIAL FLUTTER AND FIBRILLATION

Next to premature beats and the simple extrasystolic arrhythmias atrial flutter and fibrillation is the most common irregularity likely to be seen by the physician in his every day work and similar to the ectopic rhythms it may develop at all ages and in normal individuals as well as patients suffering from advanced types of cardiovascular disease The syndrome occurs in a paroxysmal form lasting for brief intervals and in a chronic form which may persist continuously over many years Only recently a group of 16 men and women at the author's cardiac clinic celebrated an anniversary marking 25 years or longer as patients with atrial fibrillation the event is noted here to emphasize that this gross disturbance of rhythm may exist as a more or less minor cardiovascular disability with minimal restrictions of activity Nearly all of the 16 persons had lived full productive years in academic and industrial areas where their individual talents and abilities were well recognized and rewarded

Atrial flutter and fibrillation have long occupied the attention of both clinical cardiologists and experimental physiologists the gross irregularity lends itself to research investigation The concept is gaining acceptance that whereas flutter may exist as a separate entity fibrillation

rarely occurs alone since flutter mechanism is continuously operative This mechanism according to Groedel Kisch Parsonnet and others is based upon the electrodynamic differences of the two atria The right atrium because of the anatomic location of the sinus nodal pacemaker remains more or less under control of its stimulus production thus it is possible for the right atrium to be in a state of flutter while the left is fibrillating In most ECG tracings showing atrial fibrillation those leads reflecting the electrical activity of the right atrium usually show the flutter complexes The former concept of circus movement as the explanation of atrial flutter and fibrillation has largely given way to the ectopic theory of multiple irritable foci arising in the atrial myocardium these productive foci may either be due to congenitally misplaced nodal pacemaker cells or certain heart muscle fibers which retain embryonal potentialities the chief of which is intrinsic stimulus production

PAROXYSMAL ATRIAL FLUTTER AND FIBRILLATION

The brief physiologic postulate presented above is important in the explanation of paroxysmal atrial flutter and fibrillation which frequently occurs in presumably normal subjects The syndrome is seen as indicated previously at all age periods the author in 1928 reported an instance in a new born infant It is possible that the condition existed *in utero*, fetal ECG s showed the irregularity during the eighth month of gestation It is seen in well conditioned athletes as well as in those with sedentary activities and it appears in equal frequency in both sexes It may follow certain indiscretions of eating and drinking smoking may be a factor especially in women

The congenital background of paroxysmal atrial flutter and fibrillation was emphasized by Miller he reported a number of cases occurring in families He was able to trace a history of sudden heart palpitations through 4 generations in the two youngest generations he established an ECG diagnosis of atrial fibrillation

Occurrence in Normal Hearts Opitz pointed out in 1941 that the normal heart never exhibits such gross disturbances of rhythm as atrial flutter and fibrillation he questioned the *normality* of those hearts where the syndrome developed He accepted the congenital concept in part but believed that some unknown alteration in atrial physiology was primarily responsible for the syndrome Sussman believed that the establishment of specific etiology has clinical significance in relation to prognosis

Do normal subjects with episodes of paroxysmal flutter and fibrillation eventually become chronic fibrillators? Parsonnet and the author in a series of 21 cases reported that only 4 became chronic sufferers of the condition other writers have placed the ratio higher Here the age incidence may have some importance with the development of arteriosclerotic heart disease there is more likelihood that the arrhythmia may become permanent Recent studies have shown that paroxysmal atrial flutter and fibrillation which develops in the later decades may become continuous after a few episodes

Symptoms Paroxysmal atrial flutter and fibrillation like the extrasystolic arrhythmias may be entirely asymptomatic more often the attack is associated with a number of sub

*jective and objective manifestations The patient may be*come aware of a sudden irregularity of the heart Pain is not a common complaint but the pounding and jumping sensation within the chest may conjure up a variety of symptomatic problems These vary from simple nausea vomiting vertigo visual disturbances throbbing in the neck and head to more severe manifestations like dyspnea weakness syncope and rarely epileptiform seizures Mild forms of pulmonary edema are occasionally seen some with hemoptysis

On auscultation the ventricular rate is usually rapid and within the range of 140 to 220 there may be a pulse deficit of 40 to 80 beats per minute In predominant atrial fibrillation the rhythm is grossly irregular but in flutter with a functioning block mechanism the rate may appear *to be* regular in the ratio range of 75 or 150 beats per minute Most episodes last for several minutes as a rule there is a spontaneous change to sinus rhythm in 5 to 30 minutes When the disturbance lasts 30 minutes or longer rales may *appear at the bases of both lungs frank pulmonary edema* may occur after several hours

The psychogenic reactions to the episode vary patients tend to respond in unpredictable patterns A previously complacent subject may become violently disturbed and may harm himself and others by irrational activity while others of more apprehensive type may remain calm and more or less unemotional during an attack Management of the greatly agitated patient may require considerable tact and understanding on the part of the physician and a clinical experience in handling the mentally disturbed will be helpful here

TREATMENT OF PAROXYSMAL ATRIAL FLUTTER AND FIBRILLATION

Since atrial physiology is responsive to autonomic and other neurogenic stimulation a number of reflex mechanisms may be employed in the attempt to convert the flutter and fibrillation syndrome back to normal sinus rhythm The usefulness of vagal reflex procedures have been demonstrated for almost 75 years eyeball pressure was described by Mueller in 1884 It still remains one of the first measures to be utilized by the physician a word of caution however should be said about the method of exerting pressure over the eyes Loewe and the author have previously reported severe damage produced in the retina and other structures of the eye by over enthusiastic manipulation of members of the intern staff in the hospital emergency room during the well intentioned attempt to stop an attack Experience has shown that the least eye injury is produced when the physician stands behind the recumbent patient and places the index and middle fingers of both hands on both eyes at the same time pressure is exerted *upward* quickly but gently Since the reflex like all other neurogenic responses is a rapid one pressure should only be applied for 3 to 5 seconds continued pressure serves no useful purpose and may place the eye in jeopardy If no change occurs in the cardiac rate or rhythm the procedure should be repeated after a lapse of at least 30 seconds No more than 4 or 5 attempts should be made by this method the rule being that it "either works or it doesn t

The carotid sinus reflex has certain advantages over eyeball pressure first the stimulation for restoration to normal

sinus rhythm is usually greater and secondly no danger from overmanipulation of the area usually occurs Here again pressure should only be exerted for a few seconds at a time repeated if necessary but never maintained continuously Overstimulation may result in complete cardiac standstill

Other vagal reflexes may be used successfully the gagging reflex is simple to produce in certain subjects Indeed the nausea which sometimes occurs in paroxysmal atrial flutter and fibrillation may thus have a teleological significance in the body s own attempt to stop the episode Patients who have experienced previous attacks of the condition learn to control an attack by a variety of methods self administered pressure over the eyeballs or over the carotid sinus is commonly practiced A finger down the throat may promote the gagging reflex forceful sneezing may end an episode in certain patients and they learn to inhale snuff effectively All of these methods bizarre as they may appear have the single purpose of producing a reflex action intense enough to promote restoration of sinus rhythm

Other Simple Measures As a rule the reflex measures are chiefly effective during the very early stages of a given attack if the episode lasts longer than 5 to 10 minutes these procedures may be worthless Therapeutic approach must be considered if the signs of pulmonary congestive failure appear most authors advise an observation period before attempting the employment of the digitalis and strophanthin preparations Inasmuch as many attacks subside spontaneously, the waiting period is in relation to the development of pulmonary edema In the interim some attention should be paid to the patient s general reactions to the syndrome The

tranquilizers and barbiturate series may be used occasionally there is need for the narcotic group codeine Demerol Pantapon Dilaudid are suggested Reducing the patient s nervous tension and anxiety may be sufficient in some instances to stop an attack without further medication

PULMONARY EDEMA

At the *first signs* of pulmonary edema more definitive therapy is indicated One of the digitalis preparations injected intramuscularly is advisable only rarely is intravenous administration required In experienced hands some of the strophanthin series are useful but the patient will demand careful observation Effectiveness of drug therapy is indicated by responsiveness of cardiac rate and rhythm it has been previously stated that spontaneous remission of paroxysmal atrial flutter and fibrillation is noted by an abrupt change to sinus rhythm The change may occur within a few beats sometimes there is an alternation of rhythms for several minutes with normal sinus rhythm finally predominating This type of restoration occurs in the reflex measures If on the other hand the rate slows down gradually from 240 to 160 or 180 for example and the rhythm remains irregular the change is probably due to the blocking effect of the drugs Ordinarily when the rate falls to with the 100 range and when pulse deficit disappears conversion to sinus rhythm takes place If conversion does not occur within 8 hours a second injection of digitoxin may be indicated

ATRIAL FLUTTER

Pure atrial flutter may present some diagnostic problems if an electrocardiograph is not available Such rhythms may

have a very high idiopathic rate in the range of 300 per minute in the beginning of an episode At this frequency physiologic block usually develops in the junctional area of the heart with the production of a 2 to 1 block and a ventricular rate of 150 An increasing resistance factor may change the ratio to 3 to 1 with a rate of 100 or 4 to 1 with a ventricular rate of 75 Unless the patient has developed subjective symptoms of the irregularity and if no telltale pulsations are noted in the great veins of the neck the syndrome may be completely missed especially at the regular 75 and 100 rate levels A simple exercise test may reveal the true mechanism present physical effort will increase normal sinus rates while it will usually have no effect on the blocked ventricular rate However exercise in certain instances may change the blocking ratio so that there may be an abrupt rise from 75 to 100 or 150 beats per minute Occasionally the reverse may take place a rate of 150 may fall to 75 or 100 after the effort test Pure flutter also responds to digitalis and strophanthin conversion to sinus rhythm may occur in a few hours after administration of the drugs

SUPPRESSIVE TREATMENT

Prevention of paroxysmal atrial flutter and fibrillation from the patient s point of view may be of more importance than stopping a given attack from previous experience he has probably developed measures to end an episode and he is vastly more interested in their prevention Very often the physician may never have an opportunity to see the individual in an actual attack the diagnosis is frequently made from the patient s history and symptoms Prevention thus

assumes a greater role in the individual s concern about his disability

If the patient presents no objective evidence of heart disease a careful review of his mode of living and correction of predisposing factors in eating drinking and smoking may be rewarding Constipation and gastrointestinal functional problems associated with gas formation should be investigated and reduced to a minimum Psychogenic problems may require a sympathetic explanation as well as the factors of unusual stress and physical strain

Where attacks continue in spite of such general health measures the employment of the suppressive drugs may be necessary As in all suppressive drug procedures regardless of the concept involved the physician is confronted with the problems of dosage and duration of treatment when to stop treatment may be just as difficult to answer as when to start (see discussion of anticoagulant therapy) As a general rule if the attacks of paroxysmal atrial flutter and fibrillation are of short duration and are controlled by the patient s own methods and if they do not recur often enough to produce either psychogenic or physical disability no other than the tranquilizers or perhaps barbiturate combinations are indicated Where however the episodes are prolonged and of frequent occurrence and especially if acute pulmonary edema is a constant threat suppressive medication is advisable

Quinidine Sulphate This has long been used for its suppressive effect in myocardial irritability leading to disturbances of rhythm it is of particular clinical value in the atrial

syndromes There is unfortunately no convenient rule of thumb in prescribing the effective suppressive dosage in any given subject, a more or less long period of trial and error may be necessary in establishing the minimum dosage required to obtain the desired results The drug is available in 3 grain tablets which may be started with one tablet 3 or 4 times a day amounts as high as 30 to 40 grains may be required in difficult cases Quinidine sensitization is not common it was seen only 4 times in over 1000 patients using the drug for one reason or another but it should be looked for in all new patients

There are a number of useful quinidine preparations Quinidine Natural (Brewer) Quindul (Brewer) Quinidine Sulphate Comp (B W & Co) and Quinidine Sulphate (Lilly) may be mentioned Quinidate—MRT (Thompson) is quinidine sulphate in an edible oil base Other esters of quinidine alkaloids have been used quinidine gluconate quinidine lactate quinidine hydrochloride and quinidine malate are available for special instances

The Procain Amide Hydrochloride Series These have also been used favorably in patients who are unable to take quinidine for one reason or another Pronestyl (Squibb) in 0 250 gm capsules taken 2 to 6 times a day as a starting dose but continued use of the drug may produce gastrointestinal symptoms

Digitalis and Strophanthin These two drugs have no place in suppressive treatment in paroxysmal atrial flutter and fibrillation occurring in normal subjects there is some experi

mental evidence that these substances may actually enhance the likelihood of such episodes occurring Both drugs should be reserved for their specific action in controlling abnormal atrial rhythms after they have developed

New drugs are constantly being discovered for the treatment of heart disease many of these are in the special field *of controlling heart muscle irritability which has practical* importance in the use of anesthetic agents Some of the new drugs show promise in both the control and suppression of paroxysmal atrial disturbances of rhythm it is possible that the next few years will see the development of a series of synthetic preparations which will make the management of these common syndromes more definitive and lasting and with them a great advancement in clinical medicine will have been made

PAROXYSMAL ATRIAL FLUTTER AND FIBRILLATION WITH HEART DISEASE

The presentation of the paroxysmal atrial arrhythmias has up to this time been chiefly concerned with the development of the syndrome in normal individuals When the condition occurs in patients with previous cardiovascular disability the outlook is far more serious When the atrial myocardium is involved in a number of pathologic processes paroxysmal atrial flutter and fibrillation is not uncommon among these conditions are myocarditis of infectious origin myocardosis or the fibrous replacement of heart muscle by scar tissue endocrine disturbances like thyrotoxicosis irreversible hypoxia as in certain forms of coronary insufficiency and cor pulmonale and a group of ill defined humoral syndromes

Pathologic Implications The appearance of paroxysmal flutter and fibrillation in any of these cardiac disabilities represents increased functional pathology with serious clinical implications the disturbance is no longer physiologic and must be considered as a part of the disease process Viewed in this light treatment of the attacks must start with a consideration of the basic pathology present and recognition of the specific causative factors responsible for the development of the condition In other words attention must be focused upon the disease process itself rather than upon the paroxysmal episode alone

Here suppressive treatment gives way to management of the predominant factors of the diseased heart as a rule improvement in the patient s general cardiovascular status is accompanied by lessening or complete elimination of the paroxysmal attacks However in long standing heart disease due to rheumatic valvular involvement hypertension or coronary insufficiency episodes of paroxysmal atrial flutter and fibrillation may usher in a permanent form of this arrhythmia After 2 or 3 attacks rarely 5 or 6 the irregularity becomes chronic and continuous

PERMANENT ATRIAL FLUTTER AND FIBRILLATION

Chronic atrial flutter and fibrillation is more common than the paroxysmal types about one third of all patients seen in large cardiac clinics exhibit this disturbance of rhythm It is seen most frequently in rheumatic heart disease but is closely followed in numbers by the arteriosclerotic group The rheumatic valvular patients constitute the largest single group with mitral stenosis predominating This statistical data is taken from a 1955 review of chronic fibrillators in the

New York area in New Orleans where rheumatic fever is less endemic arteriosclerotic and luetic heart disease leads the list It is possible that by 1965 the clinical picture will change considerably the suppression of rheumatic fever casualties by the antibiotic and cortisone preparations and the increased surgical approach in the correction of early valvular defects may reduce the number of rheumatic fibrillators below that of the coronary and arteriosclerotic groups which tend in recent studies to show an increase each year The incidence of thyrotoxic fibrillators has also diminished with the more widespread employment of the radioactive isotopes and the number of fibrillators due to acute myocarditis from various causes has shrunken under the impact of intensive antibiotic therapy It is likely therefore that the physician will see more and more chronic fibrillators of the coronary and arteriosclerotic group and less of the previous types of cardiovascular pathology This changing situation has also made itself felt in other fields of medicine and surgery text books written 10 and even 5 years ago have become antiquated such has been the rapid progress of science

Prognosis Chronic atrial fibrillation is not inconsistent with a long and useful life when ventricular rates are within the 60 to 70 range and when no pulse deficit occurs cardiac output may only be reduced 10 to 15% Many chronic fibrillators of the coronary and arteriosclerotic group require no medication congestive failure may be postponed many years by a philosophy of living which keeps the subject within his marginal reserve The "25 year anniversary class has already been mentioned and there are well documented cases who have lived as long as 50 years with this condition Compen

sated fibrillators undergo surgical procedures childbirth and other types of physical stress with a mortality no greater than certain non cardiacs of the same age group Chronic atrial fibrillation *per se* may produce less cardiovascular dis ability than a number of other heart and blood vessel diseases with greater pathologic implications

However when congestive failure appears the entire clin ical picture changes for the worse congestive failure is an index of cardiac output in which longevity is always threat ened Congestive failure may occur in an acute form or as a progressive manifestation of decompensation In emergency medicine the physician is concerned with the diagnosis and treatment of acute congestive failure in atrial fibrillation

Acute Pulmonary Edema This is common in atrial fibrilla tion *its treatment is discussed in detail in another chapter* but a few words in regard to its onset and diagnosis may be made here Acute pulmonary edema is usually the result of *sudden failure of the ventricles to maintain an adequate* and functional output such sudden failure may be due to un usual physical strain A man with a well compensated mitral *stenosis and atrial fibrillation had carried on his customary* activities as a watchmaker for about 12 years without medi cation One day he was forced to flee from a burning build ing he had to run up a fire escape to the sixth floor in order to cross over to an adjoining building When he finally reached the street he was dyspneic but able to go home by taxicab he sat in a chair for about 10 minutes and was then seized by an overwhelming attack of pulmonary edema which did not respond to emergency treatment and he died within 4 hours Acute failure may also develop during or

after general infections a woman of 38 also with mitral stenosis and fibrillation well compensated and without medication experienced a streptococcus sore throat with a rise in temperature to 102 6° F on the first day She was given appropriate antibiotic and other indicated treatment with a delayed response in the severity of the infection On the third day she suddenly went into acute pulmonary edema and in spite of intensive cardiac therapy died 12 hours later Autopsy showed extensive acute myocarditis involving both ventricles and marked dilatation of the left

Acute pulmonary edema also occurs in patients under prolonged digitalis and diuretic routines it may be difficult to explain the trigger mechanism in some in tances A lawyer age 56 with a long history of hypertension and coronary artery disease and with atrial fibrillation of 8 months standing was maintaining a more or less comfortable mode of living and working while sitting in a chair watching television one evening he suddenly went into acute pulmonary edema which was difficult to manage and which required a long period of restricted activity before he was able to return to his previous condition In retrospect what happened? While the TV program was an exciting one he had seen it before he admitted that the play had produced certain emotional reactions and that he had some heart consciousness and palpitation just before the attack

It may be pointed out in this connection that with the widespread enjoyment offered by television a new recognized hazard has been added to the list of restrictions which patients with various cardiovascular disabilities must follow Among other things television has brought to those house confined for one reason or another an amazing new world of

interest and education however the excitement and nervous *tension engendered by watching a favorite sports activity a* sentimental soap opera a tense melodrama or listening to the music of a famous symphony or orchestra may be sufficient to initiate a heart attack A number of recent reports have indicated that in responsive subjects tachycardia increase in blood pressure and emotional reaction have produced episodes of angina pectoris acute coronary occlusion cerebral accidents paroxysmal arrhythmias and acute pulmonary edema

Ventricular Rate *Acute pulmonary edema in chronic fibrillators* may occur with no or little change in ventricular rate if the rate remains within the so called *maximum capacity range* which is ordinarily from 58 to 72 beats per minute with no pulse deficit there are no therapeutic indications for more digitalis The danger of over digitalization in such instances has been previously emphasized Intravenous aminophylline *should be administered promptly atrophine with* morphine or Demerol may be necessary Oxygen should be employed and phlebotomy performed if there is evidence of considerable backward failure

Where the ventricular rate is elevated to 100 or above and a pulse deficit is present the indications for additional digitalis are more definite and one of the glucosides should be administered intravenously When the emergency measures have been successful antibiotics should be used to prevent or control the secondary pulmonary infections which tend to *follow such attacks*

VI THE PAROXYSMAL TACHYCARDIAS

One of the most dramatic of the cardiac emergencies is paroxysmal tachycardia the rapid beating of the heart in rates from 180 to 240 rarely fails to produce a number of terrifying symptoms both to the patient as well as the family The throbbing of the great vessels in the neck the visible pulsations of the heart beating against the chest wall and the synchronous shaking of the patient s body presents a charac teristic picture which has no clinical counterpart At the same time however the paroxysmal tachycardias have been called the most elusive type of cardiac emergency since many episodes are of short duration and the attack may have subsided before the arrival of the physician

Classification The paroxysmal tachycardias are convention ally divided into two groups supraventricular and ventric ular The supraventricular include those irregularities arising within the sinus nodal area from both atria from the atrio ventricular nodal area and from an ill defined junctional area which varies from patient to patient Paroxysmal atrial flutter and fibrillation have been presented in another chap ter as a separate entity since the syndrome occurs in normal subjects and is probably the most common type of tachy

cardia All other types of paroxysmal tachycardia are associated with heart disease it is rare to find these paroxysmal ventricular rhythms in individuals free from cardiovascular pathology

Definitive diagnosis of the paroxysmal tachycardias requires electrocardiographic examination and analysis occasionally a presumptive clinical diagnosis is possible Certain tachycardias may defy interpretation even after repeated ECG studies in most instances however the electrodynamic pattern can be determined by reference to standard text books on the subject Under emergency conditions an electrocardiograph may not be immediately available and the examiner will be forced to employ all his experience in inspection palpation and auscultation in attempting a diagnosis

History in the Paroxysmal Tachycardias As indicated above nearly all patients with paroxysmal tachycardia of the type under discussion will present a history and evidence of more or less long standing heart disease Hypertension and coronary artery pathology with previous myocardial infarctions lead the list in a series of cases studied at New York City Hospital individuals with arteriosclerotic disease and with normotensive pressure levels and certain post thyrotoxic patients may experience the syndrome Less common are individuals with advanced renal pathology in various grades of uremia and diabetics with vascular involvement

Telltale Premature Beats In nearly all cases there is a history of premature beats these may have occurred at intervals over a long period of months and sometimes for years The extrasystoles may have been noted as single beats or in a

series and such an ECG tracing taken at the time of the irregularity may prove to be the diagnostic clue in the interpretation of any given episode of paroxysmal tachycardia As a rule such *telltale premature beats* indicate the site of the ectopic focus responsible for the attack in a report made by Parsonnet and the author some years ago it was found that nearly 82% of the paroxysmal tachycardias under observation had shown premature beats within several months prior to the first episode and of these 94% exhibited the basic pattern of the tachycardia to be due to the same type of extrasystole It must be pointed out however that in the paroxysmal tachycardias which follow myocardial infarction new ectopic foci may develop and dominate the disturbance of rhythm In certain instances there may be multiple foci which become operative and which produce an ECG pattern difficult to analyze

Paroxysmal tachycardia may be ushered in by a number of very brief episodes lasting but a few seconds rarely as long as a minute The patient may recognize that something has happened to his heart he may describe a palpitation a throbbing or a sudden fast heart beat Occasionally there is a momentary feeling of syncope or general weakness with no specific localization Pain is uncommon but sudden dyspnea may occur with symptoms of air hunger At times the episode may be over before the individual experiences subjective feelings but ordinarily there is an awareness of sudden heart consciousness The psychogenic reactions in most patients are usually of alarm and in responsive subjects such anxiety may be sufficient to initiate another and perhaps longer lasting attack It may be this latter episode for which the physician has been called to treat and the immediate

prior history may have some significance in differential diagnosis

SUPRAVENTRICULAR PAROXYSMAL TACHYCARDIA

Atrio ventricular or nodal tachycardias follow the atrial flutter and fibrillation group in the order of frequency of the supraventricular tachycardias seen in hospital emergency service the presenting cardiac rate ranges from 178 to 240 and the rhythm is usually regular Differential diagnosis from atrial flutter can sometimes be made from the rate alone Groedel pointed out that the blocked rate of flutter followed the 150 100 75 pattern previously described in the functional ratio of atrial and ventricular beat mechanism Thus with rates above 160 with absent jugular pulsations or at a much higher rate (about 300) should suggest a nodal tachycardia However certain ventricular tachycardias may present the same rate and rhythm and final definitive diagnosis will depend on ECG examination If a previous record of the patient had shown nodal premature beats as indicated before a presumptive diagnosis may be made if no ECG equipment is available

Simple Control Measures All supraventricular tachycardias are responsive to vagal stimulation and the measures described previously may be employed as the first emergency procedure Tranquilizers barbiturate sedation and even narcotics may be necessary to control the patient's various psychogenic reactions all of which may tend to neutralize any beneficial effect to be anticipated from vagal stimulation

reflex stimulation of the accelerator mechanisms by the humoral or endocrine factors of anxiety has been emphasized by French authors Under certain conditions these factors may dominate and continue the syndrome in spite of increased vagal response

Useful Drugs To the list of drugs suggested in the control of paroxysmal atrial flutter and fibrillation may be added Neo Synephrin Hydrochloride (Winthrop) Mecholyl Bromine (Merck Sharpe & Dohme) Isuprel Hydrochloride (Winthrop) and the acetylcholine preparations Quinidine and the procaine amide hydrochloride group are less useful here

Recurrence of the supraventricular tachycardias is not uncommon and the suppressive measures discussed in flutter and fibrillation should be employed after the attack has subsided and continued for several weeks The appearance of congestive failure and pulmonary edema requires treatment previously outlined

VENTRICULAR PAROXYSMAL TACHYCARDIA

The ventricular paroxysmal tachycardias carry a more guarded prognosis than the supraventricular types this is largely due to the basic pathology usually present in most cases The syndrome is seen most frequently in hospital practice during or after myocardial infarction it is also noted in other types of heart failure and may be the terminal mechanism of death in chronic cardiovascular disease Ventricular tachycardias are usually ectopic in origin ordinarily there is one focus of irritability but occasionally 2 and sometimes 3 foci become simultaneously operative When such an ir

rritable focus arises in a functionally damaged area of the myocardium dominance of rhythm may depend more upon the electrical difference of potential developed than upon the extent of the heart muscle involved A single muscle fibre under certain physiologic conditions may produce stimuli for ventricular contraction at a greater potential and at a faster rate than those from a much larger area of myocardial dysfunction Such ectopic foci however must be differentiated from the effects produced by the *action current of injury* which occurs whenever heart muscle is irreversibly damaged this pathologic process develops in myocardial infarction secondary to coronary artery occlusion The potential difference between stimuli arising from a physiologically irritable muscle fibre and that developed by the action current of injury is said to be in the ratio of 1 to 4 or 5 Thus from a clinical point of view the paroxysmal ventricular tachycardias which occur in myocardial infarction are likely to be far more difficult to control and will have a much more serious prognosis than those developing under other cardiovascular conditions

The symptoms of ventricular tachycardia do not differ materially from those produced by the supraventricular group The sudden onset very rapid heart action, heart consciousness syncope weakness dyspnea may all be present as indicated previously, there is usually a series of short paroxysms before the major attack develops These prodromal episodes should not be overlooked in routine ECG studies taken during an acute myocardial infarct the discovery of frequent premature ventricular beats either singly or in

sequential groups of 3 to 5 may indicate the probability of a subsequent paroxysmal tachycardia and suppressive measures should be promptly instituted

Myocardial Infarction Sometimes more than one major ectopic focus develops in myocardial infarction if the area of heart muscle involvement is extensive and if both ventricles have suffered from the hypoxic pathology 2 or more points may exhibit action current of injury In such instances there may be released alternate right and left ectopic beats less common is a series of right ventricular extrasystoles followed by a series from the left It may be pointed out here that digitalis intoxication may be associated with alternate right and left premature beats in paroxysmal rhythm this syndrome may occur during myocardial infarction as the result of overdigitalization when the drug has been misused for one reason or another It may be difficult and indeed impossible to determine whether the paroxysm of ectopic ventricular alternation is due to the intrinsic electrodynamic activity of the injured heart muscle or to digitalis toxicity in certain cases There is also some evidence that injudicious employment of the drug may initiate ventricular tachycardia by increasing the irritability factors already present

Paroxysmal ventricular tachycardia occurring in the noncoronary group is far less ominous since the ectopic focus is more likely to be localized in a single muscle fibre and hence more readily controlled by suppressive measures This type is seen in the failing heart secondary to long standing rheumatic arteriosclerotic and hypertensive pathology It is also noted in cardiac patients after extensive surgical procedures

and may occur in otherwise normal individuals after certain general anesthesia administration where the diagnosis can be made by the cardiac monitor

Congestive failure and pulmonary edema develops rather rapidly after the onset of the attack ventricular output falls to 35% and less at this rate Hypotension follows with blood pressure levels in the range of 40/20 to 60/40 Effective countermeasures depend upon anticipation of the syndrome if possible Suppressive treatment after the attack has started, consists of quinidine, procaine amide preparations as well as the other drugs considered in the supraventricular tachycardias Oxygen may be necessary if cardiogenic shock is also present the treatment previously suggested for this complication should be started promptly

VENTRICULAR FIBRILLATION

The constant threat present in prolonged attacks of paroxysmal ventricular tachycardia is the development of ventricular fibrillation and functional standstill of the heart This is probably the mechanism of death in most of the tachycardias Ventricular fibrillation may also be the result of sensitivity to or overdosage of some of the very drugs employed in the management of the tachycardias digitalis strophanthin quinidine Pronestyl Neo Synephrin all produce ventricular fibrillation in experimental animals It is possible therefore that fibrillation may be drug induced in certain patients being treated for various cardiovascular disabilities

The diagnosis and treatment of ventricular fibrillation is presented in some detail in another chapter It may be repeated here however that the time factor permits no dilatory or procrastinating measures unless cerebral vascular

circulation can be restored within 7 minutes resuscitation procedures are worthless This is the most critical cardiac emergency and a knowledge of the resuscitation of the stopped heart is fundamental in understanding the complicated mechanisms at play in the attempt to restore the normal cardiac cycle The natural temptation to do *something* under the dramatic circumstances associated with functional cardiac arrest may in the long run do more harm than good unless the basic physiology is recognized

VII THE CONDUCTION DISTURBANCES

Delay in conduction time of the stimulus for contraction results in various types and degrees of heart block The blocking phenomenon may occur in the junctional areas or in the bundle system both are about equally common in clinical medicine There may also develop conduction disturbances in the terminal fibers of the Purkinje system which is sometimes seen in marked grades of bundle branch block Except for complete heart block with atrial and ventricular dissociation and in certain instances of skipped beats in incomplete heart block the definitive diagnosis is electrocardiographic

The conduction disturbances occur in a wide variety of cardiovascular disabilities, in general those pathologic processes which tend to increase the intrinsic resistance to the *passage of the electrophysical stimulus through* and along its customary pathways will produce a blocking effect Acute inflammatory reactions of the myocardium invasion of fibrous tissue into the pathways secondary to arteriosclerotic or atheromatous disease neoplasms septic foci cysts and foreign bodies are the most common basic causes

The acute inflammatory conditions include rheumatic

fever some of the virus diseases like smallpox measles scarlet fever and acute poliomyelitis and the bacterial infections like diphtheria typhoid fever and the streptococcic group associated with tonsillitis and erysipelis Other less common infections like influenza dengue and scrub typhus may also produce inflammatory myocarditis involving the conduction system It may also be noted after pneumococcus pneumonia and meningococcus meningitis and it has been reported in gonococcus septicemia and rarely in subacute bacterial endocarditis Conduction disturbances of the inflammatory type are chiefly confined to children and young adults

Sclerosing Processes In the older age groups the invasion of the conducting pathways is usually due to fibrous tissue growth and myocardial replacement it is common in arteriosclerotic heart disease and frequently follows acute coronary artery occlusion and myocardial infarction Here the disturbance in conduction is likely to be permanent and progressive compared to the inflammatory group which may return to an entirely normal ECG pattern Differential diagnosis of the etiological factors is thus of significance in relation to prognosis complete heart block for example due to acute rheumatic fever may disappear during convalescence while that secondary to myocardial infarction is ordinarily irreversible

Congenital Types Heart block both partial and complete may be congenital defects in the interventricular septum involving the Bundle of His system occur in a relatively small group of all the congenital cardiac anomalies The condition is permanent but occasionally restoration of more normal

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Prolongation of the P R interval in the arteriosclerotic group is usually permanent, the common delay is from 0 22 to 0 30 No change was seen in certain patients observed over a period of 20 to 30 years In others complete heart block developed as the sclerosing process increased this sometimes occurs within 2 to 5 years provided no coronary episodes were experienced

The emergency implications of P R delay are confined to the discovery of the condition during the acute inflammatory myocardial reactions its unexpected development during an otherwise mild general infection must be taken as an indication of cardiac involvement which cannot be ignored

INCOMPLETE HEART BLOCK WITH DROPPED BEATS

When the P R interval reaches 0 38 to 0 44' there is a tendency for an atrial beat to be blocked The syndrome may be discovered on simple auscultation the dominant rhythm is interrupted by a pause which is twice as long as the normal time limit between two second sounds heard at the apex Such intermissions in ventricular rate may be confused with premature beats with relatively long compensatory pauses in dropped beats no sound is heard during the pause while in ectopic beats the second sound of the extrasystole is usually clearly identified directly after the normal beat and before the pause

Incomplete heart block with dropped beats is seen more frequently in slow rates and since most of the acute inflammatory conditions of the heart muscle are associated with a relative sinus tachycardia the syndrome is more common in the arteriosclerotic group

Prognosis Ordinarily this type of incomplete heart block is a more or less temporary rhythm which in the older patients is a prodromal phase of complete dissociation alternations between incomplete and complete block may continue over several weeks or months before complete dissociation of atrial and ventricular rhythms is established This so called "spontaneous remission from complete to incomplete block may be misinterpreted in relation to assumed effectiveness of any given medication it is probable that the early reports on barium chloride and certain other drugs were based on failure to recognize the general tendency to remission at this stage of the blocking process

No specific medication is indicated in incomplete heart block with dropped beats the underlying and basic pathology responsible for the syndrome may require additional attention and measures should be undertaken to prevent or delay the increasing disability

COMPLETE HEART BLOCK

Complete dissociation of atrial and ventricular rhythms is one of the most dramatic discoveries in clinical medicine since its description by Stokes over 100 years ago physicians have never ceased to wonder about the mechanism responsible for independent beating of the atria and ventricles The diagnosis is ordinarily an elementary procedure requiring simple observation when the pulse rate at the wrist is 44 or less and the jugular pulsations in the neck are 72 and above and both rates are regular complete dissociation is probably present Similar findings may sometimes occur in slow coupled rhythm with alternating premature and normal beats but here the radial rate is usually one half that of the

jugular other complicated rhythms may also bear a superficial resemblance to complete heart block but these may be distinguished on auscultation

A simple test for complete heart block is the respiratory procedure of Mackenzie if there is no change in ventricular rate when atrial rate is increased on deep inspiration complete dissociation is present Certain drugs like atropine in small doses will speed up atrial rate with no change in the radial pulse rate These tests may be employed even in patients with low marginal reserve since no physical effort is required In well compensated subjects a simple exercise test may be used to accelerate the atrial rate a rise from 72 to 100 atrial contractions with no change in the radial rate is conclusive clinical evidence of the syndrome However the ECG pattern of complete dissociation is definitive no other disturbance of rhythm may be mistaken for it

The simple exercise test may also serve for another purpose increased cardiac activity may lead to a better blood supply to the ischemic area where the blocking process of the conduction disturbance has developed In some instances there may be a rise in the ventricular rate from 5 to 20 beats per minute but during the resting period the idioventricular rate usually returns to the original bradycardia In such patients the transitional stage from incomplete to complete block is probably still present and measures to promote a better coronary blood supply may be effective in postponing the syndrome

Slow Ventricular Rates When the idioventricular rate drops to 20 beats per minute the volume output of the heart has probably reached a critical level at this rate the individual

may however enjoy relative comfort if chair or bed confined Simple physical activity such as walking to the bathroom or down a hallway may be promptly followed by dyspnea and cerebral symptoms unsteadiness, syncope and complete blackouts may occur On the other hand exceptional instances have been seen where patients have carried on more or less sedentary types of work with pulse rates of 22 to 26 over several productive years A concert pianist a man of 56 with arteriosclerotic heart disease and an idioventricular rate of 26 continued to perform at a high level of perfection for almost 3 years before cardiovascular collapse Children and young adults with congenital complete heart block may perform amazing feats of physical endurance with rates of 30 to 40 one such lad observed from infancy played varsity baseball and crew with a rate of 42 with no obvious symptoms of exertional hypoxia

STOKES ADAMS SYNDROME

At lower rates than 20 and particularly in the 14 to 18 range more or less complete physical disability occurs such patients may spontaneously develop the Stokes Adams syndrome while lying quietly in bed or sitting fixed in a chair The episodes usually occur suddenly with no subjective aura or warning prodromal signs the typical Stokes Adams attack is accompanied by unconsciousness sometimes with generalized muscle twitching loss of sphincter control and with stertorous breathing There may be no radial pulse for as long as 20 to 30 seconds but regular jugular pulsations may be noted These at the time of ventricular asystole may be grossly irregular atrial flutter and fibrillation is not uncommon in this stage of complete block

The Stokes Adams syndrome may also develop at higher idioventricular rates when atrio ventricular pacemaker arrest supervenes this is somtimes seen at rates of 40 to 50 Ventricular arrest may persist as long as 40 to 90 seconds ordinarily the A V node resumes its activity spontaneously In the interval of ventricular arrest or asystole the atria continue to beat and there is experimental evidence that the accumulative effect of these blocked stimuli may be sufficient to re activate the node This type of cardiac arrest must be differentiated from total cardiac standstill since the methods of resuscitation are somewhat individualized

TREATMENT OF THE STOKES ADAMS SYNDROME

Regardless of symptoms or even in their absence when the idioventricular rate drops to the critical levels described above an attempt should be made to stimulate the A V node to greater activity a number of drugs have proved to be useful but in any given case the results are unpredictable Epinephrine has produced the most consistent favorable reaction Adrenalin Chloride (Parke Davis) in 1 500 or 1 1000 solution should be injected intramuscularly in 0 5 cc dosage this may be repeated every 20 to 30 minutes if attacks tend to recur In certain severe cases intravenous drip of 1 100 000 at the rate of 2 cc per minute may be necessary Sus Phrine (Brewer) is also useful here

Ephedrine Sulphate (B W & Co) in 25 mg doses with 100 cc of intravenous buffered solution may also increase nodal activity Paradrine Hydrobromide (S K & F) may be used

Ventricular asystole should be managed by the measures described in relation to cardiac resuscitation in this connection it may be recalled that simple thumping on the chest

wall may re activate the A V under certain favorable circum stances

Prevention of Stokes Adams attacks may be accomplished by the oral administration of Isuprel Hydrochloride Glossets (Winthrop) in 10 mg sublingual or buccal tablets every 4 to 6 hours Atropine sulphate in 1 mg dosage and homa tropine methylbromide (Novatrin) in 1/10 grain tablets may be used Ephedrine sulphate and Paredrine Hydrobromide are also effective by mouth Various other drugs have also been employed with indifferent success thyroxin Cytomel (Winthrop) Proloid (Warner) Yohimone (Breon) Pit ressin (P D Co) insulin barium hydrochloride magnesium sulphate Coramine (Ciba) and Metrazol (Knoll) may be *mentioned*

Of considerable interest are the recent studies concerning the use of the cortisone group in complete heart block with low idioventricular rates experiments have shown that in saturation and near toxic levels changes in electrolyte me tabolism with potassium reactions increase A V nodal irrita bility However the irritability factors may be elevated to levels favoring ectopic nodal rhythms so that ventricular rate may suddenly rise from 40 to 200 Potassium chloride buffered with sodium lactate solution given intravenously by the slow drip method may carry a patient over a critical period of repeated episodes of ventricular asystole

BUNDLE BRANCH BLOCK

Interventricular and bundle branch block are the result of defective conduction in the His system the diagnosis is chiefly made by ECG examination Prolongation of the

QRS interval above the acceptable normal limits of 0 10 in the conventional leads and 0 12 ′ in the precordial leads is objective evidence of conduction delay Like the atrial myo cardial conduction disturbances seen in prolongation of the P R interval any reactive process which involves the specific conducting system may increase the resistance factors in flammatory disease scar tissue toxic myocardial reactions trauma neoplasms cysts congenital defects and certain drugs produce bundle branch block patterns The lesion may be localized in the main bundle in the right or left branches or in the terminal fibers rarely all sites may be damaged

Bundle branch block is seen most commonly in arterio sclerotic and hypertensive heart disease and is chiefly a dis ability of the older age groups it is occasionally seen in children with severe rheumatic myocarditis and also in diphtheritic myocarditis It is sometimes discovered in young adults with advanced rheumatic valvular pathology In de fects of the septum either congenital or acquired as in myo cardial infarction it may be noted

Two Types Bundle branch block may be permanent or paroxysmal it is asymptomatic *per se* and no change in rhythm occurs In certain cases of advanced or long stand ing bundle branch block careful auscultation of the heart may disclose a systolic gallop which may become enhanced after a simple effort test however gallop rhythm occurs in many other cardiac conditions and if the ECG had been seen prior to auscultation iatrogenic implications are pos sible

Fluoroscopic and kymographic x ray examination may re

veal asynchronous contractions of the two ventricles in some cases this is seen most often when the QRS delay is in the range of 0 16 to 0 20 seconds

Functional Output Recent isotope and other studies have shown that there is little or no change in ventricular volume output in bundle branch block during sinus rhythm within normal rates but in sinus tachycardia of 110 and above and in atrial flutter and fibrillation there is a definite drop even with no pulse deficit Functional capacity also is diminished by bundle branch block and patients with this disability show poor physical performance curves

The emergency significance of bundle branch block is largely in relation to its clinical interpretation the discovery of the bizarre and grossly abnormal ECG pattern may lead to unwarranted prognostic implications The inverted T waves may cast a sinister shadow over the patient s activities and entire mode of living As a rule treatment and outlook should be based upon the underlying pathology responsible for the syndrome In congenital bundle branch block individuals may live a normal existence with no particular restrictions in well compensated arteriosclerotic heart disease and those with valvular defects there may be no added burdens because of the block

Increase in conduction delay may however assume some importance in coronary artery disease prior to closure and myocardial infarction an increase from 0 12 to 0 14 or 0 16 seconds over a period of weeks may indicate a rather rapid advancement of degenerative pathology If Q waves develop

simultaneously with increasing conduction delay an impending coronary episode is likely

Paroxysmal bundle branch block is usually an intermediate stage before establishment of permanent block the paroxysm may last only for a series of a few beats or it may persist for several hours or days Ordinarily the pattern becomes fixed within 2 to 6 months except in the acute inflammatory group reversion from a fixed bundle branch block to normal complexes rarely occurs Hyperpyrexia may at times permit reversion to normal conduction time a 58 year old carpenter with arteriosclerotic heart disease was found in the cardiac clinic to have a right bundle branch block pattern This pattern remained unchanged during a 2 years observation period he was admitted to the hospital with acute lobar pneumonia and a temperature of 104 4 F The fever remained high for about 5 days in spite of intensive antibiotic therapy ECGs taken on the third day showed complete disappearance of the QRS delay but on the second week the original delay pattern of 0 16 returned within 24 hours

Paroxysmal block has been discovered in otherwise normal individuals it was noted 4 times in the authors series of asymptomatic abnormal ECGs taken on Navy and Marine Corps personnel during World War II it has been found among insurance applicants and well conditioned athletes where its clinical implications may assume considerable importance Follow up of some of these individuals after a 17 year period has shown no special tendency toward the development of cardiovascular disability and it is possible that such cases may represent instances of incomplete congenital changes which become operative only under certain little understood conditions

In summary it may be repeated that bundle branch block has clinical significance only in relation to the basic and underlying pathology responsible for its production its discovery must be utilized within the framework of its physiologic implications The condition is asymptomatic and *per se* requires no treatment

VIII HEART FAILURE AND ACUTE PULMONARY EDEMA

Acute breakdown of the cardiovascular system may be ac companied by a wide variety of clinical manifestations there may be a single fleeting preagonal symptom before death or a complicated syndrome which may tax experience and patience The emergency situation may be obvious or per plexing it may require only a few comforting and reassuring words or it may demand the utmost in equipment and trained hospital personnel Acute heart failure may occur suddenly and unexpectedly in a previously asymptomatic and perhaps normal individual or it may follow a long history of many years of cardiac disability

Classification As a rule acute heart failure can be divided into three predominant clinical groups congestive failure both with and without pulmonary edema orthopnea and pain Frequently there may be a combination of any two but only rarely do the three occur together Sometimes the symptom complex presented by the patient offers no great diagnostic problem if the individual has been known to the physician and has been under his treatment a so called telephone diagnosis is possible In new patients a brief inspection and auscultation may be sufficient to institute prompt therapeutic measures Speed in the relief of the

patient s distressing symptoms is emphasized here symptomatic treatment is what the sufferer wants and needs At a later time a more complete examination if necessary may be made at a more leisurely pace and probably with better patient cooperation

Psychogenic Implications It has been pointed out before that every cardiac emergency is more or less heavily burdened with psychogenic implications this applies to the family as well as to the patient It may also involve the physician who may have to carry many responsibilities unrelated to the actual practice of medicine during the management of the case Unwarranted interference and unintentional obstruction in the performance of his duties adds nothing to the patient s welfare Emergency medicine is a special field of practice which demands quick thinking the ability to improvise and the courage to command the situation this is especially true in acute heart failure when life saving procedures must be employed and a firm policy adopted in accordance with the demands of the given clinical problem

ACUTE CONGESTIVE FAILURE

The mechanism of congestive failure has already been described and the difference between forward and backward failure has been presented in detail It may be repeated here perhaps that forward failure is chiefly due to a lessened left ventricular output while backward failure involves the concept of increased pulmonary pressure *In the beginning* the two types of failure may exist alone and predominate the clinical picture but sooner or later both are concerned in

functional cardiovascular breakdown The common denominator in congestive failure is fluid retention within the tissues and organs of the body the fluid is at first extracellular but in chronic types of edema there may also be intracellular involvement In long continued diuresis there may be considerable electrolyte disturbance sodium restriction may be accompanied by potassium deficiencies Vitamin loss is common in prolonged dehydration as well as the loss of many known and unknown important body substances Nutritional disturbances may be unrecognized in patients under prolonged chronic congestive management

Acute congestive heart failure occurs in advanced rheumatic valvular disease in hypertension in coronary artery and arteriosclerotic syndromes and in certain toxic conditions like hyperthyroidism and subacute infectious myocarditis It also develops as the result of cor pulmonale secondary to pulmonary pathology and occasionally in adhesive pericarditis In this country beriberi congestive failure is not common except in neglected individuals

Break in Therapeutic Routine Of special interest is the sudden exacerbation of congestive failure in patients under digitalis and diuretic routines this is not uncommon if a break in the treatment occurs Experience has shown that personality types play a not insignificant role here some individuals have a tendency to forget instructions while others are neglectful and haphazard in their medication Still others lean toward self medication and adjust dosage in accordance with their own whim or desire The physician will frequently be frustrated by the unrealistic attitude adopted by patients on any prolonged therapy fortunately

suitable changes in medication may be made or corrected without too much deterioration in the subject's cardiovascular status if he is seen often enough Because of the chronicity of the disability some individuals may elect to seek medical attention at infrequent intervals and months may pass without adequate supervision The acute emergency under these circumstances may require complete re evaluation of the entire status of the condition

An acute exacerbation may also occur even under strict medical observation for one reason or another the given drugs fail to produce the continued desired effects The body may develop a tolerance to digitalis for example and increased dosage may not be followed by anticipated results *Digitalis fast* syndromes are recognized in all cardiac clinics simple increase in dosage produces no change Sometimes change in the type or form of the drug given may be effective a large group of the digitalis glucosides are available and a switch from one to another may be successful Likewise the diuretics employed in maintaining an edema free state may lose their effectiveness after prolonged administration this applies more to the non mercurial group It may therefore be advisable to anticipate such conditions by alternating the type of diuretic agent every month or two The low sodium syndrome may occasionally be responsible for acute fluid retention and appropriate blood chemistry studies should be undertaken from time to time on all patients restricted to dehydration management

Acute Infections and Trauma Acute bouts of congestive failure may follow or be associated with acute infections a simple upper respiratory tract infection may be a serious

problem in chronic heart disease The virus infections are particularly prone to increase the factors leading toward acute failure Trauma of moderate grade may start an episode in a previously well controlled case a slight fall to the floor or the twisting of a joint like the ankle may precipitate an attack the shock of accidental small burns which occur during smoking certain small surgical procedures in inadequately prepared individuals have been accompanied by acute failure The list is a long and varied one and in patients with a marginal reserve such relatively minor incidents may be sufficient to turn a well controlled situation into an emergency problem

Pulmonary Edema One of the most common manifestations of an acute episode in chronic failure is pulmonary edema the clinical picture is a familiar one to nearly every physician The individual is usually seized by a distinctive type of *air hunger* all patients recognize the symptom which carries an ominous warning learned from previous experience The so-called panic reaction of Winterberg may cause the individual to perform needless activities which like a vicious circle increases the severity of the symptoms he may rush to an open window for more air or he may walk frantically around the room while attempting certain unrealistic breathing exercises This orthopneic stage was known to the older clinician as *cardiac asthma* the concept of asthma in the recent nomenclature is confined to diseases of the chest and lungs and such air hunger of cardiac origin is more accurately described as acute cardio pulmonary failure

Orthopnea This may last for several minutes and is followed by accumulation of fluid in the alveolar spaces rales appear

first at the bases of the lungs and then scattered throughout the chest When the edema fluid reaches the level of the smaller bronchi the rales become audible and constitute the rattles which are heard in this condition and which ordinarily precede the hacking cough which is characteristic The coughing may be violent and explosive in the beginning and is followed by copious sputum which becomes frothy and sometimes blood tinged Acute pulmonary edema has been called a noisy disease the harsh labored breathing the bubbling sounds of the rales and the constant rasping cough together with the moaning of the patient all produce an unforgettable clinical sound picture the diagnosis of which may sometimes be made from the next room The sufferer is usually pale and cyanotic and covered with cold clammy perspiration the facies is pathognomonic and carries the mask of fear and panic The pulse may be slow and regular but where previous irregularities have been present like atrial fibrillation the rate rises and a pulse deficit occurs There may be no or little change in blood pressure unless cardiogenic shock is present

TREATMENT OF ACUTE PULMONARY EDEMA

Morphine and Atropine These administered hypodermically have been the traditional first aid tool in the treatment of acute pulmonary edema Demerol Dilaudid and Pantapon together with atropine may be a better choice in view of the respiratory depressive effect of morphine in certain responsive patients If given promptly enough in the early phase of the attack this may be the only medication required many episodes are quickly brought under control by this simple measure alone In certain instances a second injec

tion may be necessary in 30 to 60 minutes if symptoms return

When great accumulations of fluid occur more vigorous measures are demanded intravenous aminophylline (Dubin) should be administered slowly The digitalis glucosides and Strophanthin K or Strophanthin G (Ouabain) may also be given intravenously with due recognition of the previous digitalis routine where this has been broken these drugs may produce dramatic relief but the hazard of overdosage is always present when full digitalization has preceded the attack

Phlebotomy This is indicated in obvious elevation of venous pressure usually in acute pulmonary edema due to predominant forward failure more than 500 cc must be removed to produce satisfactory results If the attack is the patient s first episode of acute pulmonary edema where phlebotomy has been indicated no great problems arise but if the individual has had a number of attacks and previous phlebotomies have been performed the question of repeated blood loss may complicate the general physical status unless a compensatory type of polycythemia has taken place The hematologic picture is further complicated by the observation that many patients treated over a prolonged interval of months with constant diuresis may develop secondary anemias and certain blood dyscrasias blood loss by emergency phlebotomy may add considerable burdens under the circumstances

Bloodless phlebotomy by tourniquet constriction of the four limbs may be useful in such instances under ideal conditions 3 blood pressure cuffs and apparatus should be employed Exact compression can be maintained with minimal

discomfort and danger from thrombosis and embolism only 3 limbs should be cut off from the circulation at any one time Compression at 10 mm Hg above systolic pressure should be applied no longer than 5 minutes and the limb not under constriction should be rotated If blood pressure cuffs are not available the physician may improvise tourniquets from rubber tubing belt straps or padded clothesline

Oxygen It has already been pointed out that oxygen is the antidote for hypoxia the air hunger and orthopnea in acute pulmonary edema is primarily due to decrease in the ventilating surface membrane of the lung by accumulated fluid rather than in one of the several disturbances of pulmonary function secondary to intrinsic membrane pathology Increase in ambient oxygen saturation does not necessarily imply greater patient absorption in pulmonary edema recent cardio pulmonary studies have shown that other factors are responsible for some of the beneficial effects observed in oxygen administration One of these factors is the *drying* or desiccation effect of pure oxygen optimum results have been obtained under positive mask pressure of 4 to 6 cm This may be difficult to maintain in the presence of continuous coughing and expectoration It is doubtful that oxygen administered either by catheter or in a tent serves any useful purpose here the psychogenic implications are however considerable and they may be used with this in mind Some patients experience immediate relief of some of their symptoms on the first inhalation of oxygen, insofar as this may contribute to the individual's comfort and peace of mind oxygen therapy will continue to have a place in the management of the emergency aspects of the syndrome

Alcohol vapor may be useful in changing the surface tension factors of the large and small bubbles of edema fluid occluding the alveolar spaces and small bronchi In clinical application pure oxygen gas from the supply tank is passed through a humidifying water bottle before patient administration this procedure should not be employed if the drying effect of the gas is needed in alcohol vapor administration this bottle may be filled with 95% ethyl alcohol and the combined gas mixture is inhaled It should be noted that alcohol oxygen mixtures of this type are very explosive it is in fact one of the rocket propellants in the guided missile field and unusual precautions must be constantly exercised during its employment in the sickroom

Anti Foaming Agents Of special interest is the development of anti foaming agents which have been taken into clinical medicine from the technical laboratories certain substances of the ethylinamine group have chemico physical properties which prevent fluid and gas mixtures from developing because of differential surface tension factors This anti bubble reaction has a number of clinical applications the most important of which is concerned with the prevention of the frothy accumulations of fluid in pulmonary edema it is probable that new drugs of this type will be available in the next few years

ACUTE PULMONARY EDEMA AND BACKWARD FAILURE

Right sided heart failure occurs in advanced mitral stenosis and in chronic pulmonary disease it is usually a manifestation of cor pulmonale Increasing resistance to right ventricular output is produced in those pathologic conditions of the

lungs associated with fibrosis emphysema bronchiectasis and pneumonoconiosis both with and without tuberculosis are the most frequent causes Here pulmonary function is disturbed by loss of ventilating area and hypoxia develops early in the syndrome Cyanosis dyspnea and increased venous pressure have been employed in the clinical estimation of the condition increase in the manifestations of these symptoms ordinarily indicates a worsening of the syndrome

Increased pulmonary pressure leads to right ventricular strain and later to dilatation functional insufficiency of the tricuspid valve is not uncommon and the phenomenon of the pulsating liver is not an infrequent discovery in more or less advanced cases The kidneys and the abdominal viscera are also involved and may complicate the cardiovascular problems by dysfunction There is usually a relatively long period of chronic backward failure before episodes of acute pulmonary edema occur this type of heart failure is less responsive to routine digitalis and diuretic therapy than forward failure Oxygen for the relief of the dyspnea and cyanosis is also less effective and aminophylline is of less value The treatment in chronic failure in cor pulmonale is far from satisfactory and many procedures may have to be tried one after another to increase the patient s comfort most of these are palliative and have no effect upon the basic pulmonary pathology

When acute pulmonary edema develops in such individuals the process is chiefly an exacerbation of the existing cardiovascular problems all of the previous symptoms of dyspnea and cyanosis are enhanced An uncontrollable cough with copious sputum occurs as a rule there is less blood streaking in this condition than in *forward failure*

The emergency measures suggested in the management of acute pulmonary edema due to forward failure may be applied here with the exception of morphine administration because of its respiratory depressive tendency Since polycythemia is not uncommon in cor pulmonale phlebotomy may be performed safely for a number of times In hospital practice this blood may be processed and re injected after its fluid content has been reduced From a physiologic stand point phlebotomy is an attempt to reduce the hypervolemia present in the backward failure of cor pulmonale if the cellular elements of the blood can be recovered and utilized the hematopoietic system will have been saved from unnecessary work

The work performance of simple respiration in pulmonary edema associated with cor pulmonale has been estimated to be one of the greatest expenditures of energy to be found in any pathologic condition the constant struggle for breath throughout a day and night cycle is one of the most distressing symptoms to be seen in clinical medicine All of the muscles of respiration—intercostal diaphragmatic and abdominal are called into play and the fatigue produced by their continuous action is frequently a major contributing factor in the acute breakdown of the failing heart From the experience gained in the use of artificial respirators in the respiratory paralysis seen in the bulbar types of poliomyelitis similar equipment may be successfully employed here and may be lifesaving in certain instances

Oxygenating by pass equipment has been a recent improvement in the surgical approach of the management of the hypoxial factors in severe cases utilization of the Bailey apparatus employed in open cardiac surgery permits blood

to be withdrawn from the great veins and re injected into the larger arteries Diminishing the strain in the heart lung circuit for as little as 20 minutes by such methods has shown much promise for extended clinical use of the method

The foregoing places emphasis upon the seriousness of this type of acute pulmonary edema, the facilities of a well equipped hospital with trained personnel may be required in some instances The physician alone and with little apparatus or supplies faces a challenging task in the management of such emergencies but not infrequently unexpected success may follow the employment of the more simple procedures described

ACUTE ORTHOPNEA AND CARDIAC ASTHMA

Acute pulmonary edema has been called a disturbance of water metabolism acute orthopnea in heart disease may be considered a disturbance of gas metabolism Orthopnea is a manifestation of hypoxia and it frequently occurs alone and without edema it may develop in normal subjects as physiologic orthopnea after exposure to strenuous physical exertion It is seen in athletes after competitive running or swimming, here the hypoxial reaction is one of oxygen debt which is quickly compensated

Orthostatic Factors Acute orthopnea in heart disease or cardiac asthma may be an orthostatic phenomenon which tends to develop when the patient is in a recumbent position it thus occurs most frequently at night when the individual is in bed asleep This type of acute orthopnea is called *paroxysmal nocturnal dyspnea* In heart failure gravitational factors tend to pool blood volume in the dependent or lower parts of

the body with the subject in a standing or sitting position such pooling will take place in the lower limbs and pelvic viscera In the horizontal position there is a shift to the lungs with increased pulmonary pressure such pressure changes may initiate a series of reflex mechanisms involving the respiratory center with the development of first simple dyspnea and then orthopnea Vital capacity is normally decreased in the recumbent position the difference between vertical and horizontal positions is about 10% In heart failure the difference may be as high as 30 to 50% Many cardiac patients are unable to lie flat in bed for this reason elevation of the head and chest by added pillows or inclined head rest is necessary in the management of this postural difference in normal breath capacity

Other factors may also play a part in the development of nocturnal dyspnea simple pooling of blood in the lungs may be a satisfactory explanation if and when the syndrome occurs every night In many patients however such an episode may occur only once in 5 to 10 days with no change in bed position Here the question of dietary indiscretions before going to bed the last smoke or nightcap of alcohol a shower bath or some type of effort or planned exercise may be the trigger mechanism Some authors believe that coitus may have provocative implications but this is not a common experience

As previously indicated the simple expedient of head and chest *elevation* may prevent many such episodes from occurring a hospital bed may be the obvious answer to the patient s problems Less satisfactory is a head rest and least of all is a number of pillows piled high Invariably there is a tendency for the patient to slip down as he turns from side

to side in his sleep when forced to awaken by orthopnea he is usually found lying more or less flat Blocks under the upper legs of the bed or a cushion placed *under* the head of the mattress with the use of only one pillow may be an expedient compromise available to the practitioner on an emergency visit

SYMPTOMS

The symptoms of paroxysmal nocturnal dyspnea are chiefly those of air hunger there is usually no physical difficulty in performing the respiratory act but the patient may complain bitterly about the inability to take a satisfying deep breath Rarely pain is present but after the attack subsides individuals frequently complain of soreness in the muscles of the chest and upper abdomen Apprehension and anxiety is always present during the first episodes but this lessens as the patient experiences a number of bouts

The attack may be over before the physician reaches the scene the ordinary uncomplicated episode lasts only from 5 to 15 minutes It may return however if the individual assumes a horizontal position again Treatment is chiefly palliative for the given attack but the underlying pathology should be studied and therapeutic preventive measures instituted Re examination of the digitalis and diuretic routine perhaps with a change of specific drug or dosage and regulation of habits may be sufficient in certain cases

The clinical importance of paroxysmal nocturnal dyspnea is in relation to acute pulmonary edema experience has shown *that such orthopnea or cardiac asthma may* be a preliminary or prodromal stage in the eventual development of pulmonary edema In this connection a word of caution must

be said concerning the general widespread use of sedatives to promote sleep in such patients insomnia is a common complaint among chronic cardiac subjects Most of them use relatively large doses of the barbiturates in one popular form or another the sleeping pill habit is unfortunately a universal problem and may complicate other drug therapy since the physician may be unaware of the situation When large enough doses have been self administered the warning reflex which awakens the patient with the onset of orthopnea may be diminished or completely lost so that the disturbance of hemodynamics eventually develops into pulmonary edema

Cardiac Asthma This may also occur during the day it is particularly common after a heavy meal Many cardiac patients have some difficulty in breathing even after a simple intake of food and drink this is especially true in obese and overweight individuals but notwithstanding their previous experiences some will continue to indulge in gourmet activities The psychogenic reaction to a diet of monotonous and tasteless food items prescribed in salt free and cholesterol low regimes may lead even the most conscientious patient to breaks in treatment One such illustration may be given since the individual in question was a 62 year old physician with hypertensive heart disease and congestive failure when he remained faithful to his restricted diet he enjoyed a more or less symptom free existence but following every banquet which he attended he was seized with a severe bout of cardiac asthma

Post exertional dyspnea or orthopnea in patients with heart disease should not be confused with cardiac asthma the oxy

gen debt resulting from physical exercise is associated with tachycardia rise in blood pressure and sometimes increase in heart size (Alsahy phenomenon) There may be little or no additional oxygen debt in cardiac asthma, ordinarily there is no change in heart rate blood pressure, or heart size determined by x ray examination during an attack Cardiac asthma must also be differentiated from certain forms of bronchial asthma where the patient s difficulty in breathing is expiratory rather than inspiratory

Heart Pain The third manifestation of acute cardiovascular failure is heart pain heart pain has already been presented in some detail but brief comment should be made here in relation to its significance in the failing heart syndrome Heart pain is uncommon in acute congestive failure and it is also infrequent in acute orthopnea or cardiac asthma it has long been a clinical observation that most patients with a history of pain lose this symptom when congestive failure develops Brooks once said that pain is an index of the functional integrity of the heart this of course is not true in cardiogenic shock where pain may be the predominant subjective symptom in more or less complete loss of cardiac function

As a rule pain is a minor complaint in the cardiac irregularities it is uncommon in atrial fibrillation during acute congestive failure and it is relatively infrequent in the paroxysmal tachycardias Subjects with complete heart block rarely have pain When pain occurs in heart failure the rhythm is usually normal more pain is noted in the slower than at the higher rates

Types of Pain Groedel differentiated the type of pain developing in compensated cardiovascular systems with that seen in the decompensated groups Wolffe following Brooks concept pointed out that a certain ill defined functional integrity of the heart was necessary in the production of the pain mechanism and that this particular state was lost or diminished in the gross irregularities of the failing heart This may be illustrated by a schoolteacher age 60 with intermittent atrial fibrillation and congestive failure the pathology was chiefly arteriosclerotic During sinus rhythm he was unable to climb a given flight of stairs in the school building without getting severe anginal episodes but when he was fibrillating he had no pain on the performance of the same exertion Another example of the difference between the pain component of compensated and decompensated individuals is the response to nitroglycerine here it has long been noted that the drug may have no or lessened effect on the pain during the failure stage whereas it produced prompt relief in the same subject when compensated

Prognostic Significance The question of heart pain during failure has a number of controversial aspects which requires further clarification but from a clinical standpoint this may be said heart pain developing during restoration of compensation *regardless of the type of cardiovascular pathology* may be considered to have a *good* rather than a sinister significance it may be a sign of improving integrity provided there is no evidence of coronary insufficiency Without defining the actual concept of "cardiac integrity within the context of its clinical implications the physician should not

be disturbed in the paradoxical discovery that certain patients experience more pain when they are fully compensated than when in failure it may be necessary in such instances to select a more or less happy medium recognizing that patient comfort may not parallel improvement in objective findings in any given case

IX THE HYPERTENSIVE CRISES

Cabot remarked that hypertension is an *instrumental disease* by this he meant that the diagnosis of the syndrome is chiefly based upon sphygmomanometric findings Today as then the discovery of elevated blood pressure levels may be purely incidental to a general physical examination in its symptom free phase it remains an iatrogenic disease In the voluminous literature on hypertension certain factual data seem clear the first is that high blood pressure is not a clinical entity Like hyperpyrexia it is a more or less constant companion of certain pathologic conditions but the relationship is not always obvious About 22 well known diseases are associated in whole or part with elevated blood pressure levels there are also 14 other ill defined conditions in which hypertension plays a role In many of these pathologic states cause and effect are still problems of research investigation as for example in renal disease

Elevation in Systolic Levels This is more common than increased diastolic pressure the ratio in cardiac clinics is about 10 to 1 A rise in diastolic pressure produces more symptoms than elevated systolic pressure it is a well recognized clinical observation that a 100% rise in systolic pressure as from

120 to 240 mm Hg may be accompanied by a few if any symptoms while an increase of only 20% as from 90 to 112 mm Hg in diastolic levels may be productive of many subjective as well as objective symptoms

The psychogenic and iatrogenic implications of hypertension may lead to more patient disability than the actual elevation of blood pressure levels Many individuals are entirely symptom free until the discovery is made, perhaps on routine physical examination life insurance examinations are a frequent beginning of *blood pressure consciousness* in responsive subjects In such patients the physician will meet with an elaborately embroidered symptomatology which may bear no relation to the type or severity of the hypertension The list of complaints given by individuals with levels of 150/90 mm Hg may exceed in length and variety those exhibited in the 250/120 range Groedel in 1940 spoke of the *hypertensive personality* this concept was unfortunately confused by later authors with the so called *high tension* individual The latter was pictured as a highly emotional type with psychiatric implications but elevated blood pressure was not included in the characterization In the original context the hypertensive personality is a patient who keys his entire philosophy of living to a real or fancied high blood pressure the physician will see a clinical difference in the two groups

THE NORMAL LIMITS OF BLOOD PRESSURE

At the turn of the century, Osler observed that normal systolic levels were approximately the subject's age plus 100 this formula has also been attributed to several other writers The concept was used as a rule of thumb for almost 30

years it was then generally abandoned in favor of actuarial data obtained in life insurance studies where the limits of 138/88 mm Hg were established by mutual acceptance for candidates of 21 and older Normal levels were said to average 120/70 in a review of 10 000 applicants who received ordinary life coverage Many clinicians have believed these levels are far too low in view of the longevity statistics of individuals rejected for blood pressure levels above these figures Recent investigations by Master Garfield and Walters *indicate* that *average* or normal blood pressure findings are much higher in the general population than that employed by life insurance groups and that as a rule systolic blood pressure increases as age advances A number of mathematical formulae have been developed in the past 15 years based upon military statistical data the Bøe Hummerfelt Wedervang formula for example is widely used in Europe In this country there has been a tendency to utilize the simple Osler formula again in the calculation of so-called normal levels in view of the importance of age factor For practical purposes age plus 100 is satisfactory up to the age group of 60 in older groups blood pressure levels become lower in normal" individuals

Diastolic levels at 100 mm Hg and above are of clinical significance regardless of age period life expectancy is definitely shortened in subjects with permanent or fixed high diastolic levels A blood pressure finding of 240/90 other factors being equal is more likely to have far less prognostic implications than 180/110 mm Hg Diastolic levels are generally more static than systolic following the standard exercise tolerance tests for example systolic levels always show a greater variability in comparison with diastolic pressure

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treatment for such elevated levels under the circumstances is a difficult clinical problem

At a recent symposium on hypertension it was the unanimous opinion of the panel experts that asymptomatic hypertension with diastolic levels of 90 mm Hg and below requires no treatment regardless of the systolic elevation this rule however imposes certain obligations upon the physician With systolic levels at 200 mm Hg and above frequent examinations are required under such a concept here *frequent* implies at least once a month or 6 weeks Increase in heart size by x ray study changes in the ECGs significant urinary findings retinal involvement are all objective evidence that the high systolic levels are exacting a penalty even though no subjective complaints are elicited On the other hand and this is a common experience many such individuals show no clinical reaction after many months and years of careful observation in some cases the elevated systolic pressure completely subsides after a given period of time although there may be a number of remissions over the years

Where there is an associated rise in diastolic pressure and regardless of negative clinical findings the indications for suppressive therapy are clear sooner or later such asymptomatic patients present the complications of hypertensive cardiovascular disease and delay in treatment becomes hazardous notwithstanding possible psychogenic implications

SYMPTOMATIC HYPERTENSION

It has already been pointed out that there may be a marked lack of correlation between subjective complaints and objective findings in any given hypertensive subject the same lack

of correlation may occur between symptoms and the degree or extent of the hypertension Some clinical appraisal is necessary in every case prior to any type of management estimation of the psychogenic factors is equally as important as the determination of blood chemistry renal function heart size and ECG studies The patient should thus receive the benefit of such psychiatric examination and treatment as may lie within the scope of the physician himself except in extreme instances It is doubtful that many such individuals should be referred to psychiatric specialists experience has shown that more eventual harm than good is usually the result of the implied significance of psychiatric consultation both to the patient as well as to his family *Every physician is qualified to look into possible emotional conflicts which may have a bearing upon the patient s blood pressure a sympathetic and understanding approach in this area may sometimes be more rewarding than the entire physical examination*

Use of Tranquilizers In this connection a preliminary course of moderate tranquilizer therapy may produce unexpected results in many instances systolic levels may fall to within more or less normal limits A recent case illustrates the importance of stripping off the emotiogenic factors a lawyer newly appointed to his first high corporation assignment was seen because of headaches dizziness palpitation and insomnia *His blood pressure was found to vary from 208/98 to 230/104 mm Hg* After 2 weeks control with one of the meprobamate preparations his pressure fell to 160/88 mm Hg with almost complete relief from his subjective complaints Since no other medication was employed this case also illustrates the difficulty of assessing the effectiveness of

the hypotensive drugs which also carry certain psychogenic favorable implications

Many of the available hypotensive drugs recognize the need of removing this emotiogenic factor before the intro duction of the tranquilizer group the various barbiturate drugs were used in combination with the vasodilator sub stances Theominal (Winthrop) Diurbital (Amfre Grant), Thesodate (Brewer) and other similar preparations have been useful but in individuals sensitive to barbiturate after effects the tranquilizer comb nations like Miltrate (Wal lace) are preferred

The emotiogenic factors have less importance in certain types of advanced hypertensive disease with more or less extensive kidney and cardiac damage here the more thera peutically potent drugs have a definitive role The Rauwolfia substances the hexamethonium series and the conjugated sulphur drugs like Diuril (Merck Sharpe & Dohme) Diamox (Lederle) and Esidrix (Ciba) are indicated

The surgical approach to the management of hypertension which does not yield to drug therapy and other measures has received increasing attention in the past 15 years The physi cian should be familiar with the scope and limitations of these procedures which may offer some promise of increased life expectancy in malignant high blood pressure regardless of the mechanism or etiology involved in any given case Cau tion must of course be exercised in the selection of individ uals for the various types of surgery which may be employed not all patients receive benefits commensurate with the many post operative complications which tend to occur Moreover in many operated cases a return to the previous high pres sure levels is not uncommon On the other hand where pa

tients have been carefully studied in the light of the various hypertensive surgical classifications results have been gratifying

THE HYPERTENSIVE EMERGENCIES

Patients with both increased systolic and diastolic blood pressure levels may live and enjoy many activities for a number of years after the onset of the syndrome sooner or later however physiologic breakdown occurs in three vulnerable areas brain heart and kidney Usually one of these areas bears the brunt of the pathology occasionally two or the whole three may be equally involved The breakdown is ordinarily a slow but progressive change suffered by these organs with secondary complications resulting from their dysfunction The basic pathology is a vascular reaction to the pressure factors with the terminal arteriolar system showing the primary disturbances this is followed by the secondary or correlated alterations in the specific tissues supplied by these vessels At this stage most of these changes are irreversible and simple lowering of the pressure levels is not necessarily followed by improvement in the patient s condition In certain instances there may be a spontaneous drop in pressure leaving the individual with the problems of advanced renal or myocardial failure or with cerebral involvement A paradoxical situation sometimes occurs in which such lowered pressure levels may actually enhance the symptomatology of the diseased areas

Each of the involved organs may show signs of acute failure during the terminal phase of the hypertensive syndrome the mortality rates are about equal in the three major areas Possibility of survival is lessened when more than one area

has reached a certain critical stage but the proper manage ment of the emergency may add weeks and perhaps months in any given case The emergencies engendered here are fre quently responsive to definitive treatment and in spite of the advanced pathology recovery from the immediate crisis is possible and may be unexpectedly gratifying As in all life saving measures the physician has a duty to perform which does not recognize the probability of defeat

THE HYPERTENSIVE CARDIAC EMERGENCIES

It has been noted previously that enlargement of the heart is a common finding in advanced hypertensive disease the en largement is first localized on the left but eventually both ventricles are about equally involved The initial cardio megaly is due to hypertrophy of the musculature but dilata tion occurs as the pathology progresses The x ray silhouette is characteristic with symmetrical increase in both contours It is doubtful that any marked degree of failure occurs in simple hypertrophy failure is ordinarily the result of dilata tion

There is apparently no consistent correlation between hy pertension and ventricular output increased systolic pres sure does not necessarily indicate increased output In some instances measured output is less under the conditions of elevated systolic pressure than when more normal levels are present Increase in the size of the heart likewise may bear no relationship to output When dilatation occurs output falls this may take place with no obvious change in blood pressure

Forward failure is primarily the result of lessened ventricu lar output it is associated with terminal arteriolar dysfunc

tion and the accumulation of extracellular fluid Gravitational factors play a selective role here dependent parts of the body become edematous Both the liver and the kidneys may be involved in the electrolyte disturbance which develops during hemodynamic failure and which leads to fluid retention in the body tissues Forward failure in hypertension is rarely an acute phenomenon it may require many days before large accumulations of fluid become evident

Backward failure in hypertension on the other hand may occur rather rapidly acute pulmonary edema can develop in minutes Backward failure can arise from a sudden fall in right ventricular output secondary to the strain factors associated with dilatation of the left ventricle A less acute form of pulmonary edema takes place in functional insufficiency of the mitral valve when the valve ring is stretched *in dilatation of the left ventricular musculature the valve* leaflets become incompetent and whole or partial systolic intraventricular pressure may be transmitted backward through the left atrium to the pulmonary veins Increase in pulmonary pressure under such changed physiologic conditions may be promptly followed by pulmonary edema Pulmonary edema causes a marked decrease in vital capacity with an accompanying increase in hypoxia Lethal hypoxia is the primary cause of death in acute pulmonary edema

Emergency treatment of such acute pulmonary edema is concerned with both sides of the heart functionally there is too much blood on the venous side and too little on the arterial *The hypervolemia of the right heart is much easier* to remedy than the hypovolemia of the left phlebotomy is indicated with the gradual withdrawal of 300 to 500 cc of

blood Strict asepsis should be maintained in such removal the blood may be used later as in cardiogenic shock for intra arterial injection It should be emphasized at this time that phlebotomy should not be performed *per se* in the attempt to control or lessen high systolic blood pressure this formerly held concept is no longer tenable in view of the Naval Medical School research projects which showed in 1950 that the physiologic mechanism of blood pressure control produces an elevation in pressure gradients when blood loss occurs Experiments in normal subjects show a systolic rise in consistent relationship to reduction in blood volume until a critical level of hypovolemia develops at which point hemorrhagic shock takes place This reflex may be paradoxically enhanced in hypertensive disease so that it is not uncommon to note an actual rise in systolic pressure during and after a phlebotomy of 500 to 1000 cc In one such patient an immediate increase from 248/115 to 262/120 mm Hg was discovered after removal of 450 cc of blood about 3 hours later the pressure fell to 200/104 mm Hg

Under favorable circumstances reduction in venous pressure may be promptly followed by marked improvement in the patient s orthopnea but relief may be only temporary unless a better left ventricular output can be obtained As earlier indicated intravenous digitalis medication may be lifesaving a variety of the digitalis glucosides are available for this purpose Digifolin (Ciba) Digiglusin (Lilly) Digitaline Nativelle (Varick) Gitaligin (White) Lanoxin (B W Co) Purogigin (Wyeth) Crystoldigin (Lilly) as well as the several preparations of digitoxin or digoxin may be employed with equally satisfactory results

Digitalis Complications It may be wise to repeat here that some problem of overdosage may occur if the patient has been on a prolonged digitalis schedule in urban practice it is unusual to find an individual with acute congestive failure due to hypertension who has not had previous digitalis medication of some type The dosage may have been irregular or of subclinical value in certain instances patients become *digitalis fast* a condition similar to but not identical with drug tolerance The question of digitalis storage is important in this connection it is a well recognized clinical experience that rapid diuresis in such patients may be accompanied by symptoms of overdosage and digitalis intoxication because of the sudden release of huge amounts of the drug from the liver

Where acute pulmonary edema has occurred in a hypertensive individual who also has marked fluid retention for example with great edema of both legs ascites hepatomegaly and other evidences of general anasarca and where there has been a relatively long administration of digitalis caution must be exercised in the emergency addition of the drug in the attempt to control the situation Here carefully employed intravenous mercurial diuretics given together with aminophylline may serve a two fold purpose first by their combined fluid clearance effect and secondly by potentiating the stored digitalis Under certain conditions there may be a decided improvement in the pulmonary edema within 30 to 60 minutes

A word should be said about the inherent hazards of digitalis intoxication in this type of pulmonary edema The development of premature beats with conduction disturbances are of more clinical significance than the appearance of ST

segmental and T wave changes although all of these are ECG evidence of myocardial toxicity When ectopic foci occur in both ventricles the likelihood of paroxysmal ventricular tachycardia is greatly enhanced and the step between this rhythm and ventricular fibrillation may be very small There is unfortunately no specific antidote for over digitalization but some drugs may tend to neutralize the more dangerous toxic effects of the drug Atropine alone or in combination with procaine amide hydrochloride (Pronestyl) may sometimes be effective Insulin in shock doses has been recommended by recent German investigators on the concept that hypoglycemia lessens myocardial irritability under certain conditions Hypothermia studies carried out in connection with open heart surgery have shown that hypertonicity of the heart muscle due to a number of causes is considerably reduced after intravenous injection of cold solutions All of these measures are heroic attempts to combat lethal types of cardiac arrhythmia which must be considered by the physician under such emergency circumstances

Atrial Fibrillation This is less frequently seen in acute pulmonary edema associated with hypertensive disease than that due to rheumatic valvular syndromes mitral stenosis is a common factor in the latter conditions Here the associated cor pulmonale produces a different type of pulmonary edema in which backward failure is the predominating disturbance in the hemodynamic mechanism

THE HYPERTENSIVE CEREBRAL EMERGENCIES

Next to acute congestive failure the acute cerebral reactions to elevated blood pressure levels constitute the largest group of emergency problems caused by or associated with hypertension The chief pathologic changes are concerned with various grades of increased intracranial pressure as a simple complication of elevated blood pressure levels and increased intracranial pressure due either to arterial occlusion with infarction or vascular rupture with hemorrhage Increased spasticity of the smaller arteries of the brain is common in hypertension there is still considerable debate concerning the mechanism involved Its clinical importance is in relation to the differential diagnosis of so called *small strokes* due to such vascular spasm and those due to emboli or thrombosis

Simple increase in intracranial pressure of the type seen in hypertension is probably a manifestation of cerebral edema since edema of the brain is rarely seen in congestive failure other factors appear to be involved in its production Small to moderate degrees of cerebral edema may take place over a period of days or weeks with few if any symptoms but sudden increases in intracranial pressure above a critical level produce a number of dramatic and alarming reactions

Acute Hypertensive Encephalopathy This usually occurs in patients with a long history of markedy elevated blood pressure levels which range in the 260 to 300 systolic and 140 to 160 mm Hg diastolic group it is relatively uncommon in levels of 220/110 mm Hg and below It is not seen in acute pulmonary edema but it may occur in renal breakdown where the cerebral symptoms may be confused with uremia

Symptoms Subjectively the individual complains of head ache localized in many instances to the occipital area the headache may be mild or of unbearable severity Dizziness and vertigo may be present in varying degrees nausea and vomiting is not uncommon Visual disturbances may develop blurring of vision blind spots and sometimes diplopia may be described In severe cases coma and unconsciousness de velop quickly Objectively the patient presents a flushed face irregularly dilated pupils and sometimes twitching of the muscles of the face and neck Visible pulsations of the temporal arteries and throbbing of the great vessels of the neck are common the head may nod with each pulsation The pulse is of full volume usually regular but occasionally premature beats may be noted The neck is tender on mo tion and if the patient is semiconscious there may be marked rigidity of the cervical and sometimes the upper shoulder muscles The superficial reflexes may be exaggerated and rarely there is a unilateral difference The respirations are slow but regular even in profound coma the Cheyne Stokes syndrome may not occur

The emergency treatment of acute hypertensive encephalop athy is concerned with the possible measures of reducing the cerebral pressure factors A drop in systolic blood pres sure may not be followed by relief of intracranial symptoms Phlebotomy in certain instances may enhance the cerebral pressure factors in accordance with the physiologic reflex previously discussed in which systolic pressure rises imme diately after blood volume loss This reflex may however be diminished or suppressed during coma or partial uncon sciousness experience has shown that when venesection is

performed slowly i e at a rate of 10 cc per minute, a fall in systolic pressure may take place after the removal of 400 to 500 cc It is important in this connection to point out that a relatively small decrease in pressure may be accompanied by marked improvement in the cerebral disturbances, in one patient a drop from 268/158 mm Hg to 245/146 after the withdrawal of only 280 cc produced a change from unconsciousness in a 65 year old woman to a more or less comfortable condition with minimal headache and vertigo On the other hand rapid removal of 750 to 1000 cc has not changed the clinical picture materially in other cases

Lumbar Puncture This is indicated in most patients with acute hypertensive encephalopathy spinal tap is not always a simple procedure in agitated or unconscious subjects It is best performed with the patient lying on the right side, complete exposure of the area is necessary and the physician will require some assistance in maintaining the proper position The needle is inserted by the conventional method and intraspinal fluid pressure is quickly determined by the *drop count* normal drop count is about 1 per second for the first 20 seconds depending upon the needle gauge Ten to 15 drops indicates greatly increased spinal fluid tension actual spurting occurs in the dangerously elevated cerebrospinal pressure syndromes

Arterial blood pressure determinations should always be made simultaneously with spinal fluid tap occasionally there is a fall in systolic pressure with removal of less than 2 or 3 cc of spinal fluid If the fall is greater than 40 to 50 mm Hg the lumbar puncture should be stopped irreversible shock

phenomenon may occur in such rapid decrease in blood pressure levels A drop from 296/158 to 180/110 in a patient after removal of 12 cc of fluid under great pressure produced a state of shock which nearly led to a fatal outcome in a recent hospital emergency room instance Several small withdrawals of fluid under pressure (about 4 to 8 cc) over a period of one hour may be more successful in producing a sustained drop in blood pressure than a single large tap

Spinal fluid in acute hypertensive encephalopathy is usually clear and free from macroscopic blood a sanguineous tap after the first 3 or 4 cc have been removed suggests hemorrhage in the cerebrospinal system and the presumptive diagnosis of simple hypertensive encephalopathy is in question All neurologists believe that a *bloody tap* requires further study and examination since the removal of such sanguineous spinal fluid may do more eventual harm than good under the circumstances the lumbar puncture should be stopped

Spinal Anesthesia If the removal of spinal fluid under pressure is not accompanied by symptomatic relief of the acute encephalopathic syndrome intraspinal anesthesia may be attempted operating room experience has shown that patients with high blood pressure are particularly responsive to the hypotensive factors associated with routine spinal anesthesia Only 1 ml of the standard anesthetic dosage should be administered under careful blood pressure observation a second 1 ml may sometimes be necessary A number of anesthetic preparations are available Blockain Hydrochloride (Breon) Cyclaine (Merck) Novocaine (Winthrop) Nuper

caine Hydrochloride (Ciba) Procaine Hydrochloride (Harvey) and Xylocaine Hydrochloride (Astra) may be mentioned

Hypotensive Reactions Occasionally, there may be a delay of 20 to 45 minutes after spinal injection before full hypotensive reaction develops if there is a marked drop in blood pressure approaching critical shock levels countermeasures may be necessary to combat the clinical overdosage Here Coramine (Ciba) Metrazol (Knoll) Mikedimide (Panray) and Wyamine sulphate (Wyeth) may be employed cautiously and with due attention to both the cardiac and respiratory systems

Acute hypertensive encephalopathy was formerly considered to be a *pre apoplectic* state by many of the older clinicians recent studies however have shown that less than 35% of all cerebral hemorrhages are preceded or ushered in by this syndrome When death occurs in this type of encephalopathy the mechanism is chiefly due to disturbance of brain stem function with both respiratory paralysis and cardiac standstill Post mortem examination usually reveals the brain to have been under great pressure miliary punctate hemorrhages may cover the entire cortex as well as involving the brain substance throughout Massive hemorrhage does not occur except in the basilar area in a few cases

Patients ordinarily experience several episodes of acute hypertensive encephalopathy there may be complete recovery with no loss in mental or neurologic capacity As a rule each succeeding attack becomes more difficult to treat and the outlook becomes guarded after the second or third epi

sode Under a routine or plan of strict preventative measures however some patients may live to within their normal life expectancy

CEREBRAL HEMORRHAGE

It has already been pointed out that about 66% of all strokes due to cerebral hemorrhage are not preceded by acute hyper tensive encephalopathy vascular rupture in the brain is usu ally a sudden episode with no or very few prodromal symp toms Such symptoms would vary depending upon the size of the damaged artery its location in the brain and finally the most important factor the amount or volume of extrava sated blood Patients who have recovered from such attacks state that they either had no warning prior to losing con sciousness or they may have experienced a peculiar sensation in the head difficult to describe pain is apparently not a pre dominant symptom compared to a feeling of sudden confu sion and disorientation *sometimes accompanied by abrupt* aphasia Not all vascular ruptures are associated with uncon sciousness a hemiplegia may occur in bed while the patient is asleep Such patients awake in the morning to find one side completely or partially paralyzed they may be unable to speak but are more or less well oriented

The familiar capsular hemorrhage with rupture of the *middle or other striate arteries is usually* associated with com plete unconsciousness for several days in the less severe cases The statistical data here show that about 18% die in the first 8 hours while 28% have a fatal termination in 24 hours A better outlook for survival occurs if the individual passes the first 5 days death after the first week or 10 days is chiefly

due to a number of complicating factors Pneumonia ne phritis and anuria secondary to prostatic involvement and certain general infections are common

EMERGENCY TREATMENT OF ACUTE CEREBRAL HEMORRHAGE

The indications for definitive treatment in cerebral hemor rhage are simple stop the bleeding and remove the extra vasated blood clot However since these procedures are problems of brain surgery which must be performed within a short period after the onset of the episode the opportunity for such an approach in the management of the syndrome remains somewhat limited in scope in the hands of compe tent brain surgeons and under optimum circumstances a number of successful cases have been reported Subdural and basal hemorrhages present a far less difficult situation than capsular injuries the trauma secondary to the vessel rupture and the surgical approach usually leave irreversible brain damage but improvements in technique and manage ment will probably lessen these complications

Where surgery is contraindicated or can not be performed for one reason or another the physician has a number of emergency measures which may ameliorate in some degree the possible consequences of cerebral hemorrhage Since the episode is primarily due to the hypertensive syndrome an immediate drop in systolic pressure may lessen the volume of extravasated blood All of the methods employed in com batting the acute encephalopathic problems may be well utilized here Phlebotomy must be attempted with all of the precautions previously described Arterial puncture with the removal of 500 to 750 cc of blood may be effective in limit ing the extent and pressure factors of the cerebral accumu

lation both of the clot and the secondary reactive edema Careful spinal puncture may be employed to reduce cerebral and spinal pressure spinal anesthesia is not recommended in the presence of acute brain damage although the procedure has been successful in the British Navy Simple measures like hypothermia of the head may have some symptomatic value

Pulmonary Complications Of paramount importance is the maintenance of a clean and free airway to the lungs all of the accepted methods used by anesthetists in providing adequate respiratory function may be employed This may imply just a simple adjustment of the jaw or the introduction of an airway tube in certain instances it may be necessary to perform a lifesaving tracheotomy Inspiratory pneumonia is the rule in any unconscious subject in cerebral hemorrhage antibiotic therapy should be started promptly

Renal Problems In men and sometimes in women there is usually loss of sphincter control of the bladder at first there is enuresis followed later by retention An indwelling catheter of suitable size should be inserted within 6 hours after the onset of unconsciousness and left in for several days it may be advisable to continue catheter management for sometime after the patient has recovered orientation Scrupulous asepsis is mandatory it has already been noted that one of the fatal complications in this syndrome is irreversible kidney damage

Fecal incontinence may also occur directly after the stroke a small cleansing enema should be given to prepare the rectum and colon for possible injection treatment Satu

rated magnesium sulphate solution in 100 to 250 ml volume has certain recognized clinical value in reducing cerebral pressure due to various causes it may have a limited place in cerebral hemorrhage Saturated sodium phosphate may also be used for this purpose

Dehydration This is common in strokes due to cerebral hemorrhage there is always a great water loss secondary to sweating vomiting and rectal incontinence Since intravenous infusions are contraindicated because of the extravasation factors of the bleeding artery it is advisable to administer such replacement fluid by rectum after the magnesium sulphate procedure has been completed The basic water requirements of such unconscious patients is about 1800 to 2000 cc per 24 hours

Hyperpyrexia In varying degrees hyperpyrexia occurs during the first several days after such a cerebral accident the usual rise is to about 100° F Occasionally fevers in the range of 104 to 106° F may occur in rare instances 108 and even 110° have been noted Temperatures over 105 usually terminate fatally it may be a manifestation of extensive brain damage Recent advances in the use of controlled hypothermia in cardiac surgery makes available these measures and apparatus in the management of such types of excessive fever In a well equipped hospital hypothermic methods may also be useful in the routine treatment of all cerebral accidents there is accumulating clinical evidence that patients so treated may have a generally better outlook

Diet No attempt to *feed* the patient should be made for at least the first 24 hours it should be emphasized again *that*

inspiratory pneumonia is a constant threat in unconscious individuals Even washing the mouth and tongue with bland solutions may cause foreign material to be inhaled into the bronchi and lungs It is also doubtful that there is any gastrointestinal function during the initial period of the attack attempts to instill water or nutrient fluids by gastric intubation may lead to gas formation and dilatation of the stomach A certain degree of gaseous distention always accompanies most cerebral accidents this may be due to vagal stimulation Elevation of the diaphragm reduces vital capacity and contributes to the patient s respiratory problems

Tube feeding may be started carefully on the second day accumulations of gas should be removed before the nutrient material is administered There may be considerable retention of gastric contents from the previous meal eaten by the individual since cerebral hemorrhage is not uncommon after a heavy meal the postprandial stomach may contain a large residue of undigested food which was not removed by the vomiting experienced during the early phase of the episode This is a frequent autopsy finding and the volume of such retained food may be amazing in a recent case nearly 2 liters were measured As much of this undigested residue as possible should be aspirated from the stomach and a tube of sufficient calibre should be employed

Arterial rupture in hypertension is a manifestation of vascular disease experiments by Buerger and the author in 1927 showed that normal arteries may sustain a simulated systolic pressure as high as 500 to 750 mm Hg without tearing of the vessel wall Atheromatous and arteriosclerotic pathology is chiefly responsible for weakening of the arterial wall in extensively involved vessels rupture may occur at

autopsy at relatively low blood pressures sometimes at levels much below that found during life A typical instance was that of a woman age 74 who suffered a cerebral hemorrhage with a blood pressure of 188/118 mm Hg a dissected tibial artery tore under a simulated systolic pressure of only 156 *mm Hg*

Mycotic aneurysms and certain specific diseases of the arteries may lay the groundwork for spontaneous rupture even at normotensive levels While relatively uncommon these syndromes must be borne in mind when for example hemiplegia develops in individuals with more or less normal blood pressure

If there are obvious signs of vascular pathology it is likely that the heart is also involved chronic heart strain and coronary artery pathology are usually present There may be a history of one or more cardiac episodes prior to the cerebral accident It has been pointed out that cerebral hemorrhage is uncommon during congestive failure likewise coronary artery occlusion with myocardial infarction is rare during cerebral hemorrhage The heart itself presents no immediate problems during such a stroke episode and usually no specific *treatment is indicated Some cardiologists* believe that under the circumstances a decompensated cardiovascular system is to be preferred on the concept that the factors enhancing the hypertensive syndrome may be lessened during the critical period of arterial rupture and bleeding into the brain substance

CEREBRAL THROMBOSIS AND EMBOLISM

Although most cerebral vascular accidents associated with high blood pressure are related to arterial rupture and hem

orrhage some mention of other types of vascular episodes should be made Cerebral thrombosis and embolism both with and without infarction occasionally occur in hypertensive patients but the syndromes are far more common in normotensive individuals as well as in low blood pressure subjects Thrombosis of the cerebral arteries is also a manifestation of atheromatous and arteriosclerotic vascular disease the fragmentation and separation of such murantic thrombi is productive of free floating emboli with subsequent occlusion of vessels distal to the original vascular lesion The symptoms of cerebral embolism will vary of course depending upon the size of the vessel obstructed and its anatomic location in the brain occlusion of one of the major arteries to the so called *silent areas* of the frontal lobes may exhibit few or no signs in comparison to a relatively tiny embolus in the capsular area with its associated hemiplegic syndrome

The differential diagnosis between vascular rupture with hemorrhage and vascular occlusion with infarction may be difficult in the early stage of the episode unconsciousness may occur in both as well as paralytic symptoms If the patient is normotensive and if there is obvious atheromatous pathology present the most likely diagnosis is thrombosis or embolism This is particularly true if there is a rapid recovery from the neurologic component of the attack Recent consensus among clinicians is that *small strokes* are thrombotic or embolic in origin the former concept of spastic contraction of cerebral arteries has given way to the occlusive mechanism in view of post mortem discovery of such arterial pathology in patients who have had a history of two or more small strokes

Prognosis Almost complete recovery is the rule in cerebral embolism unless a major irreversible neurologic disaster has occurred restoration of function may start in the first 24 hours Extensive thrombosis requires a longer period for functional restoration Both of these conditions stand in contrast to the markedly delayed recovery when rupture has taken place the difference is due to the specific pathology involved Thrombosis and embolism are *intravascular* accidents while rupture produces *extravascular* change The irreversible damage caused by extravasation of blood into the brain substance is usually far greater than the functional and temporary infarction secondary to occlusion

TREATMENT OF ACUTE CEREBRAL EMBOLISM AND THROMBOSIS

The emergency treatment of acute cerebral embolism and thrombosis is more or less similar to that discussed in hemorrhage, particularly if unconsciousness has occurred The measures required for reduction in blood pressure unless extremely high, may not be necessary Likewise, lumbar puncture may not be required except as a diagnostic procedure

Embolism ordinarily produces sudden and acute cerebral symptoms in comparison to thrombosis which may be a relatively slow process with a more or less prolonged prodromal phase during which time the patient may exhibit a variety of reactions Headaches sometimes unilateral dizziness mental confusion nausea aphasia and certain neurologic complaints of a sensory type like tingling numbness and burning sensation of the skin in different parts of the body may occur for some minutes or hours prior to the major episode

A number of drugs have been suggested for use at this stage the entire vasodilator group papaverine aminophylline nitroglycerine caffeine and the niacin preparations administered intravenously may be effective in limiting the secondary reactions The anticoagulant drugs present a challenge in treatment here the original concept of Wright s anticoagulant therapy both for the prevention and treatment of cerebral thrombosis and embolism has been shown in several recent reports to have resulted in more brain damage than that produced by the episode itself This is especially true if there has been a misdiagnosis of thrombosis for example when arterial rupture has actually taken place here heparin and the dihoxicoumadin drugs enhance the extravasation process by delaying the normal clotting factors However there may be a place for the heparin preparations where there is a tendency for repeated attacks of thrombosis or embolism to occur over a period of several days Of paradoxical interest in this connection is the reported use of the Vitamin K series by English clinicians where small hemorrhages are suspected in hypertension these drugs have been given in combination with rutin and ascorbic compounds

Extracerebral thrombosis with cerebral embolism is relatively uncommon such emboli usually arise in the heart and are the result of fragmentation of murantic thrombi which develop in the left atrium or ventricle secondary to myocardial infarction In mitral stenosis during paroxysmal atrial fibrillation emboli may be released from thrombus formations in the left auricular appendage Unless there is a coexisting open foramen ovale or a ventricular septal defect emboli arising in all other parts of the body are filtered out in the pulmonary circulation fat emboli from fractures air

emboli from improper intravenous procedures and septic emboli from thrombophlebitis of the pelvis or lower legs rarely reach the brain for this reason However where cerebral emboli are known to be of cardiac origin anticoagulant therapy is definitely indicated regardless of other considerations

The discovery of thrombolytic substances has opened a new concept in the treatment of general thrombus formation with special reference to cerebral thrombosis while still largely in the experimental stage much promise of the development of a new series of synthetic preparations useful in the control of thrombosis and embolism has been given by research workers in this area of preventive medicine

SMALL STROKES

A final word may be said about the concept of small strokes since their recognition may have some bearing upon the prognosis of the hypertensive syndromes Alvarez has defined a small stroke as a fleeting glimpse into the future welfare of the patient he might have added that from small strokes large ones may promptly develop

Symptomatology It has been said that the symptomatology of small strokes is remarkably protean in that a multitude of minor and otherwise insignificant subjective and objective complaints may develop The chief of these are sudden and temporary aphasia or amnesia giddiness without postural change or physical effort fleeting visual disturbances usually of both eyes sudden incoordination particular of the hands and fingers when engaged in some delicately adjusted procedure like writing or sewing and sudden dif

ficulty in walking or running Sensory disturbances include tingling or numbness in one hand or arm or on the side of the face or head rarely are the subjective symptoms painful The entire episode may last but a second or two rarely as long as a minute

Unless the syndrome is repeated a number of times the individual may entirely forget the incident in responsive patients the first attack may bring them promptly to the physician s attention When such a history is given by a subject of the middle age or older groups a thorough physical examination is mandatory In previously normotensive individuals this may be an early sign of cerebral vascular pathology in those with a known history of hypertension regardless of the actual levels *energetic treatment is indicated* as well as a change in the routine of daily living which may be enhancing the progress of the given pathologic process

Prognosis Recognition of the small stroke pattern thus has a number of clinical implications beyond the immediate emergency situation its development in hypertensive patients should be viewed with some prognostic significance regardless of the apparent fleeting disability of the condition at the moment and the tendency perhaps of the individual to minimize its occurrence It may prove to be an only clue to a larger and more serious problem in the near future

X CARDIAC RESUSCITATION

The greatest cardiovascular emergency is cardiac standstill with breakdown of the physiologic mechanisms responsible for the sequential phenomena of the beating heart Unless some volume of ventricular output is maintained lethal hypoxia promptly develops it has been estimated that survival is possible even when per minute volume has been reduced to 10% of normal In complete heart block with a rate as low as 2 beats per minute recovery has taken place and in total cardiac arrest resuscitation may occur after 5 to 7 minutes of ventricular standstill

Hypoxial Resistance There is considerable variation in the resistance to maximum hypoxia exhibited by the important organs of the body the brain is the most responsive to oxygen debt The Stokes Adams syndrome is a clinical manifestation of cerebral hypoxia when ventricular rate drops to 10 or 15 contractions per minute in complete heart block or in any other type of abnormal bradycardia the motor areas of the brain may exhibit characteristic symptoms The sensory areas may however develop certain reactions much earlier with rates of 20 to 30, coma and unconsciousness have been noted even in rates of 40 to 44 *There is no consistent corre*

lation between minimal ventricular output and the type of cerebral hypoxial reaction patients may be mentally alert and responsive to stimuli at extremely low levels of arterial competency Under the same conditions of hemodynamic physiology others are in deep coma and may show characteristic pre agonal symptoms

Subminimal Bradycardia Complete cardiac arrest must be differentiated from *subminimal bradycardia* subminimal bradycardia has been defined as the slowest possible ventricular rate consistent with peripheral vascular circulation As indicated above such rates may be as low as 2 to 3 beats per minute rarely 1 beat or 1 every 90 seconds In these instances the mechanism responsible for the cardiac cycle while more or less grossly abnormal is still functioning and therapeutic measures designed to increase ventricular output by increasing rate involves a vastly different concept than that concerned with cardiac standstill There has been a recent tendency to include such stimulating procedures under the consideration of cardiac resuscitation which by accepted definition is the attempt to reactivate a heart which has completely lost its functional integrity

Two major types of cardiac arrest are recognized clinically complete standstill of the entire heart and ventricular fibrillation The differentiation is important and has many implications both as to treatment and possible survival as well as in the management of the post resuscitation syndromes

COMPLETE STANDSTILL OF THE HEART

The normal cardiac cycle is dependent upon the rhythmic activity of the sinus nodal pacemaker the conductivity of

the atrial myocardium the responsiveness of the atrio ventricular node the transmission of the impulse through the bundle system the integrity of the Purkinje network and finally the reaction of the receptor mechanisms of the ventricular musculature Failure at any point in this delicately adjusted physiologic circuit results in certain disturbances of rhythm and conduction which are responsible for the various cardiac irregularities noted in the electrocardiogram The most important disturbance of the cardiac cycle is failure of the sinus nodal pacemaker to initiate the electrochemical stimulus upon which the entire cycle depends without such a stimulus there can be no ventricular contraction and hence no output from the heart To mitigate and guard against the possibility of such a hazardous disturbance the atrio ventricular node retains a variable embryonal capacity to initiate its own stimulus for myocardial contraction this type of functional response is seen in complete heart block with its characteristic slow idioventricular rate It is also found in hyperactivity of the A V nodal mechanism with release of premature beats here the rate may be very rapid as in paroxysmal nodal tachycardia when such beats dominate the rhythm of the heart

A V Node Potentialities The so called *standby* functional capacity of the A V node is apparently potentiated by a number of complicated physiologic factors in assuming its lifesaving activity as in complete heart block there is a relatively long period of adjustment which usually precedes this dominant role in the maintenance of the cardiac cycle On the other hand sudden failure of the sinus node to initiate a stimulus may not find the A V node ready to take

over pacemaker function and as a result of which complete cardiac arrest occurs As indicated above one of the important factors here is concerned with the retention of specific cell mechanisms which promptly come into play on sinus nodal failure this emergency functional capacity varies from subject to subject and is lost in the advancing age periods presumably due to increasing myofibrotic changes

Complete cardiac arrest from pacemaker failure is more common in normal hearts than in patients with cardio vascular disease it is far more frequent in children than in adults It occurs frequently during anesthesia procedures it is more likely to occur during inhalation methods than in intravenous types of general anesthesia Psychogenic stimuli may be a cause for sinus nodal block it is probably the mechanism by which responsive individuals are frightened to death by excessive vagal stimulation It may also result from certain neurogenic trauma sudden death from the so called solar plexus punch was said by Stern to be due to complete cardiac arrest in boxers and wrestlers Specific out lawed judo maneuvers where intense pressure over both vagus nerves in the neck are used have caused complete cardiac standstill Death by hanging is primarily due to asphyxia but Cannon reported that cardiac arrest may occur first because of vagal effects

Stages of Hypoxia Standstill of the heart is immediately fol lowed by three recognized stages of hypoxia The first stage which lasts about 4 minutes is characterized by a rapid rise in the irritability factors of the ischemic myocardium a complicated series of metabolite changes produce extra cellular differences of electric potential which only require a

trigger mechanism to develop a stimulus for myocardial contraction At this moment a simple thump on the chest wall over the precordium may initiate a parasystolic ventricular contraction of sufficient volume output of blood to flood the coronary arterial system In many instances activation of one or both nodes occurs with prompt return of the cardiac cycle in a more or less normal pattern

Intracardial Injection If simple percussion or compression of the chest wall does not produce immediate results intracardial injection procedures are indicated no time should be lost at this critical point in myocardial hypoxia A variety of substances have been used in intracardial injection but experimental and clinical evidence indicates that the needle prick itself may be sufficient to reactivate the stopped heart The *action current of injury* produced by the needle thrust becomes a focus from which ectopic contractions may develop Under favorable circumstances a single ventricular contraction may be followed by restoration of sinus activity but as the interval of hypoxia increases this focus may continue to liberate a rapid series of stimuli at a rate potentiated only by the refractory phase of muscle response This rate may be as high as 400 to 480

From a physiologic standpoint the anatomic location of the needle thrust is of considerable importance if the intracardial injection has been made in the ventricular areas a grossly abnormal rhythm similar to paroxysmal ventricular tachycardia may develop with all of the potentially hazardous sequellae which occur in such rhythms and which will be discussed later under the problem of ventricular flutter and fibrillation *When on the other hand the injection is*

made in the atrial areas the same physiologic response will take place but since rhythms like atrial flutter and fibrillation are well known clinical entities which are responsive to appropriate treatment a much more favorable outcome is possible Intracardial injection should therefore be made in the atrial areas during this phase of attempted cardiac resuscitation

If the simple needle thrust fails to reactivate the cardiac cycle injection procedures should now be employed as indicated previously a large number of drugs have been used more or less successfully for this purpose and there is no general agreement concerning the effectiveness of any given substance Adrenalin in 1 100 dilution has been the choice in some schools of thought atropine lidocaine ether alcohol and pituitrin have been used Even distilled water has given a favorable reaction in certain cases

The Second Stage of Myocardial Hypoxia This develops about 4 minutes after cardiac standstill this stage is characterized by an increasing loss of muscle response to stimuli regardless of intrinsic or ectopic origin It is still possible however to reactivate important physiologic factors of contraction provided the coronary system can be restored to minimal functional capacity Such restoration may be produced by manual contraction of the ventricles this procedure implies an open approach to the heart and the employment of a special technique which will be described subsequently In non cardiac patients resuscitation by surgical methods has taken place for as long as 12 minutes after cardiac arrest but ordinarily the chances of survival diminish rapidly after 6 or 7 minutes This possible survival period coincides with

the limits of reversible brain damage patients who have successfully made a cardiac recovery after a prolonged second stage of hypoxia usually suffer from more or less extensive cerebral involvement

The Third Stage of Myocardial Hypoxia This stage is only of academic interest, it occurs after the 7 minute period perhaps somewhat longer in certain instances In addition to irreversible brain damage intravascular clotting both arterial and venous may have taken place thrombosis and embolism may be extensive with involvement of various organs and limbs In unsuccessful attempts at manual procedures in the second phase autopsy examination has shown widespread embolization and peripheral thrombosis which would have made eventual recovery unlikely even though reactivation of the heart had occurred

It should be repeated here that complete cardiac standstill due to pacemaker block regardless of the mechanism responsible for this type of cardiac arrest is more responsive to resuscitation measures than any other cause for this emergency syndrome In patients free from heart disease it is probably the most common clinical type of cardiac arrest and the physician should be familiar with the basic physiologic data concerned with the development of the condition He should also understand the significance of the prompt life saving procedures which should be undertaken in the management of this otherwise fatal disturbance of cardiac rhythm

FUNCTIONAL CARDIAC ARREST

Cardiac Output It has been previously emphasized that no cardiac output is possible without functional ventricular

contraction *Functional ventricular contraction* may be defined as that type of ventricular contraction or systole which develops a hemodynamic pressure gradient high enough to open the aortic valves against a residual diastolic pressure level thus permitting a flow of blood into the arterial tree In the normal cardiac cycle *no* blood leaves the left ventricle until diastolic pressure resistance above the aortic valves is more than equalized In other words functional ventricular pressure must always be greater than diastolic pressure regardless of the gradient level at the given instant in the systolic period when diastolic pressure becomes greater the valves close and the blood remaining in the ventricle becomes the *sub diastolic pool* As noted previously in the well conditioned athlete this pool may be equal to 40% of total ventricular capacity

When abnormal rhythms develop the pressure gradient factors may be grossly disturbed in premature beats for example the stimulus for contraction may occur before sufficient blood has accumulated in the left ventricle to permit the equalizing pressure to open the valves A pulse deficit thus occurs this hemodynamic phenomenon also is seen clinically in certain types of atrial fibrillation In the more rapid arrhythmias like paroxysmal tachycardia volume output is limited by the same mechanism The menace of the ectopic ventricular tachycardias is primarily loss in ventricular output although the heart as a whole may be exhibiting wild activity

In ventricular fibrillation there is no coordinated contraction of the ventricular musculature as in atrial fibrillation the rapid twitching of the muscle fibers serves no functional purpose and insofar as the cardiovascular system is con-

cerned cardiac arrest has occurred Functional cardiac standstill in contrast to arrest due to pacemaker block is usually a disturbance of a diseased heart it is seen most frequently in coronary heart disease but it also occurs in acute myocarditis secondary to a number of viral and bacterial infections It may be the mechanism of sudden death in uremia diabetic acidosis and in certain overwhelming endocrine disturbances like thyrotoxicosis acute hyperadrenalism, and hyperinsulinism It may also develop in trauma to the thorax and may be the immediate cause of death in electrocution although there is some evidence in this latter condition that if the pathway of the current has not been through the heart or mediastinum pacemaker block may have occurred secondary to intense vagal stimulation, it may be of some importance in such cases to determine the exact pathway of the electric current in the body

In the functional cardiac arrest due to ventricular fibrillation the three stages of hypoxia previously described are not clearly defined The fibrillary movements of the heart muscle produce a number of electrolyte changes which tend to enhance or neutralize each other in a series of grossly abnormal physiologic processes The responsiveness to ectopic stimuli seen in complete cardiac arrest is lost in ventricular fibrillation and intracardial injection procedures are usually fruitless unless the fibrillation is terminated

Control of Ventricular Fibrillation A number of methods have been suggested in the management of ventricular fibrillation the author s method is perhaps as useful as the more compli

cated measures which require equipment not generally avail able to the physician at the time of this extreme emergency condition Experiments have shown that ventricular fibrilla tion which develops in the heart free from cardiovascular pathology may be suppressed by intra pericardial flooding with drugs of the procaine series procaine amide hydro chloride (Pronestyl) lidocaine hydrochloride (Xylocaine) and procaine amide glucomate (Plungianate) have been used successfully In practice about 50 to 75 cc of the given solution is injected into the pericardial cavity near the ap proximate location of the apex of the heart neither the needle point nor the solution should be injected *into* the heart muscle or ventricular cavity In fortunate cases fibril lation may stop promptly but subsequent intracardial injec tion may be required to reactivate the cycle

Survival Rhythm Unlike the complete recovery which may occur in the normal heart after successful restoration of physiologic cardiac rhythm following pacemaker block res toration of *survival rhythm* in ventricular fibrillation is lim ited by the basic pathology responsible for the episode of cardiac arrest Treatment of the fibrillation leaves untouched the abnormal syndrome which produce the trigger mecha nism and recurrence is likely to happen If for example the fibrillation was initiated by a myocardial infarct secondary to coronary artery occlusion restoration of functional ventricu lar output mav be temporary and fleeting On the other hand there are many reports of survival after such attacks of fibril lation and in spite of the discouraging overall statistics one should proceed with his utmost skill and perseverance in the

attempt to resuscitate each patient thus stricken One successful outcome in this desperate emergency will outweigh any number of failures

OPEN CARDIAC RESUSCITATION

The surgical approach to reactivation of the stopped heart has gained more or less wide acceptance in the past decade great advances made in cardiac surgery have opened up new methods of resuscitation Indeed pocket knife surgery" has been successfully accomplished even by the informed laity and there are increasing daily press accounts of such life saving attempts by non medical personnel

While a pocket knife has a traditional role in emergency surgery there are other instruments which are better adapted for exposure of the heart and the physician must have a place in his professional bag for them side by side with his usual syringes and needles they constitute the minimum armamentarium which he must carry with him on every emergency call Cardiac surgeons recommend at least 4 instruments these are a sharp scalpel, medium size straight scissors, a curved snap and a pronged forceps

A number of surgical procedures have been described in exposure of the heart in cardiac arrest but the quickest method is through the diaphragm from the left upper abdominal quadrant Intercostal incision and rib spreading is far more difficult in adults and elderly patients more instruments are required and the method should be reserved for cardiac standstill occurring in or near an operating room Surgery in the stopped heart syndrome has been compared to post mortem manipulation there is no arterial bleeding but there may be considerable venous oozing An incision

is made just below the left costal margin observing aseptic technique insofar as this may be possible under the given circumstances since time is of the very essence only those parts of the subject s clothing should be removed to permit a satisfactory exposure of the chest and abdomen The exploring hand is thrust through the abdominal incision and the left leaf of the diaphragm above the left lobe of the liver which should not be injured A second incision is made with the scissors through the diaphragm into the pericardium If possible the left pleural cavity should not be invaded since collapse of the lung increases the problems of cardiac massage The heart should be gently and carefully palpated sometimes the mere opening of the pericardial cavity and touching of the myocardium may be sufficient to reactivate the stopped heart (see previous note on ectopic stimulation during the first phase of hypoxia) A differential diagnosis between complete standstill and fibrillation is usually not difficult to make the violent twitching movements in fibrillation have been compared to the fluttering of butterfly wings in a closed hand while in complete arrest no movement of any kind can be determined Occasionally the movements in fibrillation are so vigorous that the examiner may be startled by the activity of the heart in a presumably dead patient

Effective manual massage of the heart requires some dexterity the exploring hand should be placed as high as possible in the pericardium so that the apex of the heart lies over the physician s wrist In this position the palm of the hand is posterior to the left ventricle the thumb and little finger are slowly brought together *with the palm and not the fingers* exerting pressure There are many reports in

medical literature concerning damage to the myocardium produced by pinching or squeezing the heart with the fingers Broad surface pressure is far more effective in raising intraventricular pressure sufficient to open the aortic valves

Manual massage of the stopped heart is preferably a two-man job there is need for an observer as well as an operator The functional effectiveness of *artificial ventricular contraction* is determined by the appearance of a palpable beat in one of the peripheral arteries Clinical experiments have shown that unless a pulsation can be identified at the radial or temporal artery with each compression of the heart the *procedure is functionally worthless The primary objective* of resuscitation of the stopped heart is restoration of coronary blood flow the pressure gradient required for the lessening of myocardial hypoxia is equal to the pressure necessary to produce a pulse wave in an artery about 18 inches from the aortic valve

Manual compression of the heart should be performed gently and slowly rapid squeezing and pinching as indicated before may do more harm than good The optimum rate is from 30 to 40 compressions per minute In cardiac arrest *the artificial diastolic filling period requires* a much longer time interval for the accumulation of blood in the left ventricle than when the normal cycle *is* operative The right ventricle and pulmonary function are also at a standstill and unless blood can be drained from the pulmonary *pool none can be forced from the left side* of the heart

The heart can also be exposed by incision through the chest wall this may be the incision of choice in the operating room when cardiac arrest has occurred during surgery This is a much more difficult approach when the emergency

takes place elsewhere outside of a hospital or clinic In children and young adults rib spreading and sternal mobili zation can sometimes be performed with limited instrumen tation, the patient is placed on his right side and the usual anterior axillary incision is made in the 4th or 5th inter costal space In adults and elderly subjects with a rigid rib cage such an exposure presents a number of technical prob lems with the available emergency instruments

If successful resuscitation has resulted from any given procedure the patient should of course be rushed to the nearest hospital for surgical and shock treatment No at tempt should be made to close wounds at the scene of the emergency The danger of infection, secondary hemorrhage embolization and the recurrence of cardiac arrest demands the attention of a competent hospital team When cardiac function returns the subsequent after care is better left in the hands of surgeons and under scientifically controlled conditions

THE ARTIFICIAL PACEMAKER

It is just 29 years since the author developed the first appara tus for resuscitation of the stopped heart It was chiefly based upon the postulate supported by experimental and clinical evidence that in intracardial injection the mecha nism of reactivation of the stopped heart was due to an ectopic focus produced by the action current of injury dur ing the first and second stages of myocardial hypoxia This physiological phenomenon has already been described in detail Since the origin of this ectopic focus may be unde pendable and once started may be difficult or impossible to control the concept was developed at the Witkin Founda

tion for the Study and Prevention of Heart Disease Division of Beth David Hospital New York about 1928 that if a simulated pacemaker current within the normal range of 0 1 millivolt was passed through a special type of insulated needle and liberated at a point at or near the sinus nodal area of the right atrium such a stimulus might follow the normal conduction pathways in the heart with sequential contraction of the various cardiac chambers The rate of contraction could be controlled within the range of the given age group

The artificial pacemaker as its name implies thus attempted to substitute for the sinus nodal pacemaker when dysfunction of the latter lead to cardiac arrest The instrument over the years has been continuously modified and recent models are about the size of a pocket flashlight and may be easily carried in an emergency bag It is a lifesaving piece of equipment when used in complete cardiac standstill it is less effective in cardiac arrest due to ventricular fibrillation

Resuscitation apparatus for the control of ventricular fibrillation is chiefly based upon the concept of *countershock* Whereas the artificial pacemaker applies a direct electric current within the normal electro physiologic range of the human heart (0 1 to 0 5 millivolts) countershock equipment utilizes a high voltage alternating current (55 to 220 volts) The basic theory here is dependent upon the maximal response concept in *the stimulation of muscle tissue i e* at any given instant following the refractory period of contraction muscle response will result from the maximum stimulus when more than one stimulus is present

Counter shock apparatus is standard equipment in most operating rooms several types are available They are all more or less complicated devices and rather expensive and not usually within the scope of the internist s armamentarium However the theory of countershock occupies a definite role in emergency medicine where cardiac arrest due to ventricular fibrillation has been diagnosed The cautious application of ordinary 110 volt AC house current has been successfully employed in a number of reported instances The procedure demands rigid compliance with certain fundamental observations

Improvised Countershock Methods If previous methods of resuscitation have failed and if for one reason or another open massage of the heart can not be performed the patient is placed upon a wooden floor or carefully insulated from all metallic surfaces by *dry* rugs or blankets A nearby extension cord is removed from its wall outlet and both wires are cut at a point sufficiently long to reach the patient the insulation at the cut ends is removed leaving at least 2 inches of exposed wire One should wear dry gloves (preferably rubber) when handling the exposed ends during the countershock procedure The electric plug is inserted into the outlet when all is ready and with caution the two exposed wires are placed in the left and right axillae for a second and then removed The heart should be auscultated and the radial artery palpated to determine possible heart action This procedure should be repeated if necessary with a 2 second period of stimulation and if required a 3rd or even 5 attempts should be made increasing by 1 second the period of each succeed

ing application It should be emphasized that no ausculta tion or touching of the patient by any one should be made *while the electric current is in contact with the body*

While application of the electrodes at the axillae has been the most successful in this simple type of countershock ex periments have shown that with the patient on the right side the electric current may be applied to the front and back of the chest It may also be applied to the palms of both hands or the left hand and precordial area The electrodes should be kept away from the face and neck

It should be repeated again that countershock by this method may be rewarding but it also may be of some hazard to the physician as well as to those standing near the patient Emphasis has been placed upon *dryness* such attempts at resuscitation should never be made in wet surroundings as at or near a swimming pool a wet bathroom or in a similar environment As a strictly emergency measure and under the circumstances described it may in certain otherwise doomed patients be of lifesaving potentialities

ARTIFICIAL RESPIRATION

Cardiac arrest may or may not be simultaneously associated with recession of respiration In complete cardiac standstill respiration may continue for several minutes while in ven tricular fibrillation breathing stops more rapidly In cardiac resuscitation pulmonary function plays an important role since the basic physiologic disturbance in cardiac standstill is the development of increasing hypoxia the ventilating cycle demands as equal attention as the heart during all life saving procedures If there has been a surgical approach in the attempt to reactivate the heart it is likely that spontane

ous collapse of the left lung has occurred in more or less extensive degree it may be impossible to expose a given heart without opening the left plural cavity Reduction in more than 60% of available vital capacity may seriously jeopardize pulmonary function by increasing the hypoxial syndrome cycle

Artificial respiration must thus be maintained throughout the resuscitation procedures and the physician should be familiar with the acceptable methods of providing for adequate pulmonary function during the cardiac emergency period Since the usual methods of artificial respiration utilizing compression movements of the thorax can not be employed while management of the stopped heart are under way mouth to mouth respiration is the most satisfactory no apparatus is required and any bystander may be quickly instructed in the procedure In children direct mouth to mouth artificial respiration presents no difficulties but there may be obvious objections in adults and in the elderly Here a suitable airway may be quickly constructed from rubber or other tubing a piece of garden hose a short length of water pipe or even a spout from a water kettle has been improvised for this purpose More satisfactory of course are the various types of airways employed in the operating room most of these are small enough to be included in the emergency bag

The rate of artificial respiration like that of manual massage of the heart should be slow and regular there is always a tendency to perform all manipulations too rapidly under the excitement of lifesaving circumstances Mouth to mouth respiration is more effective at a rate of 10 times per minute than at one of 20 more harm than good may occur in exchange rates of 25 and above especially in children It may

be necessary to continue artificial respiration for relatively long periods sometimes for as long as 40 minutes or an hour Actual experience with mouth to mouth respiration has shown that a well conditioned athlete may perform this service for about 10 to 15 minutes without fatigue but in those less physically fit 2 or 3 minutes may be the limit of their capacity It is therefore desirable to have a number of volunteers take over this important duty In a recent instance of mouth to mouth artificial respiration 6 persons carried on for almost 2 hours with a successful outcome

Volume of Air Some attention should be paid to the volume of air forced into the patient this is of special importance in children where rupture of the lung has occurred from alveolar over distension There is a tendency to take as deep a breath as possible before blowing the air into the airway depending upon the size and age of the patient normal vital capacity should be estimated and carefully adhered to by each volunteer

Negative Pressure A word should also be said about the negative phase of mouth to mouth respiration the suction effect should be carefully determined Blood streaked mucus is usually a sign of too much negative pressure although some bleeding always occurs during this type of artificial respiration The negative pressure cycle should be somewhat longer than the positive and should not be performed until there has been at least a 3 second pause rapid in and out breathing should be avoided

Oxygen is of little practical value here, unless the individual performing the procedure takes a deep breath of 100"

oxygen before blowing into the recipient s lungs this is a complicated maneuver but it has been attempted successfully a number of times with experienced personnel If pressure breathing apparatus is available it may be alternated with mouth to mouth respiration

POST CARDIAC RESUSCITATION TREATMENT

It has been noted previously that complete cardiac arrest due to pacemaker dysfunction usually occurs in individuals with more or less normal hearts when reactivation has developed promptly there are ordinarily few symptoms of cardiovascular or pulmonary complications However even in cardiac standstill of short periods there may be some in volvement of the lungs with rales and small areas of atelecta sis *The patient may show a slight rise in temperature in* 24 hours and a cough may develop It is advisable in all such cases to start antibiotic therapy on admission to the hopsital

Inspiratory Pneumonia If there has been a prolonged period of mouth to mouth artificial respiration considerable pul monary trauma may have taken place this may vary from simple alveolar distention to rupture of small and large areas of the lung partial or complete collapse of one or several lobes as well as bronchial laceration Infection is always present inspiration of foreign body material from the donor s mouth is the rule A septic type of pneumonia with antibiotic resistant staphylococcus strains is not an uncommon sequella In all instances of successful cardiac resuscitation the chest should be repeatedly x rayed to determine the extent of the pathology present Such pulmonary disease may persist for several weeks or months An illustrative case is that of a 54

year old truck driver upon whom a successful reactivation of the stopped heart was performed by intracardial injection of adrenalin and simple mouth to mouth respiration for only 4 minutes he developed a series of pulmonary abscesses a staphylococcic empyema which required surgical intervention, and a purulent bronchitis which demanded almost a year of constant treatment

The post resuscitation period in ventricular fibrillation usually presents a complicated clinical problem since many of these patients have a more or less prolonged history of heart disease the basic pathology responsible for the episode of fibrillation remains unchanged or worsens as the result of the development of cardiac arrest Here one is confronted with a double threat i e, management of the pre existing disease as well as the trauma resulting from the resuscitation procedures both in the heart and the lung Ordinarily there is a stormy recovery period which will tax the utmost skill of the physician with death hovering over the bedside from any one of several possible complications Experience has shown however that if the patient can be carried over the first 10 days eventual recovery to *status quo ante* is not uncommon It is interesting to note in this connection that relatively few cases have suffered a new coronary attack as the result of the cardiac arrest death usually occurs from intractable congestive failure or from irreversible pulmonary complications

The cerebral aspects of the post resuscitation syndrome are chiefly neurologic certain psychiatric complications sometimes occur in greater or lesser degree Their consideration has no place in a presentation of the emergency implications of cardiac arrest but a word should be said about the

renal disturbances which may follow standstill of the heart in periods longer than 4 minutes Acute hypoxia of the kidney with its associated ischemic phenomena may produce irreversible changes in its functional capacity azotemia may quickly occur Urinary suppression is not uncommon and uremia may be difficult to control The urine should be promptly examined after all resuscitation procedures and appropriate treatment should be anticipated

In brief the complications following a successful restoration of cardiovascular function after standstill of the heart may produce many diverse and perplexing clinical problems In the general relief and satisfaction enjoyed by all engaged in such lifesaving measures little attention may be paid to the possible and likely sequelae which may follow It need not be emphasized that cardiac arrest is a serious disability which is not ended by reactivation of the heart in every case there is to be expected one or more complications which may have untoward implications and which will require a period of prolonged medical care

ANTICOAGULANT THERAPY

Thrombosis and Embolism As indicated previously, the post resuscitation syndromes following cardiac arrest have not received the attention deserved by these complications in deed these complications have frequently rendered the entire procedure worthless as well as throwing doubt upon the wisdom of treating cardiac standstill at all It has been pointed out that the second stage of hypoxia which extends up to about 7 minutes after cardiac arrest coincides with the period beyond which irreversible cerebral damage takes place It also more or less coincides with the onset of intra

vascular clotting with the development of free floating thrombi One of the chief hazards after reactivation of the cardiac cycle is thus embolization

Medical literature of the past decade is replete with instances of successful resuscitation followed by eventual death of the patient from massive infarction of the brain kidneys *lungs as well as occlusion of the vessels of the limbs and* mesentery Usually, fatal thrombosis and embolization occurs within 24 to 48 hours Occasionally such infarctions do not appear for several days a patient well on the road to recovery may be suddenly stricken with a series of overwhelming emboli

The problem of preventing such intravascular coagulation *must be seriously considered in every case of successful re*suscitation as a general rule the longer the cardiac arrest has occurred with its associated circulatory standstill the more likely thrombus formation has taken place If there has been a prompt response in reactivating the stopped heart and a period no longer than 90 seconds has occurred the factors of subsequent embolization are much less than after a 7 minute period of circulatory collapse

Some authors have questioned the value of anticoagulants here to be effective the drug must be given in large enough dosage to produce a prompt effect in clotting time In resuscitation methods which have required an open exposure of the heart the danger of hemorrhage is constantly present There is however less validity in this concept when reactivation has occurred without the employment of surgery

The heparin series of substances are the drugs of choice they act quickly and are usually readily controlled by the Vitamin K_1 preparations The general use and control of the

anticoagulants has been discussed and the same measures may be used in the post resuscitation period Heparin should be employed for at least 72 hours it may be discontinued after the oral drugs have produced a satisfactory prothrombin level

As in myocardial infarction no rule may be layed down concerning the time for withdrawal of the anticoagulants If there has been no clinical evidence of embolization a month s treatment may be sufficient with gradual lowering of the prothrombin time to normal levels If however a series of embolic complications have taken place it may be necessary to continue such therapy for several months Most observers favor Sussman s concept that anticoagulant therapy must continue for at least 3 weeks after the last embolic manifestation

DIAGNOSIS OF CARDIAC ARREST

In the conventional presentation of clinical material a discussion of differential diagnosis ordinarily precedes the problems of treatment In this chapter diagnosis has purposely been kept for the end until the mechanism of cardiac standstill is well understood and the response to its management is presented in detail the major points in differential diagnosis can not be clearly delineated With this in mind the questions involved in the diagnosis of the stopped heart may now be discussed

Under operating room conditions where cardiac monitors and other special equipment are available there may be no problem in making a prompt diagnosis of the stopped heart Ventricular fibrillation is easily recognized and complete standstill due to pacemaker block can be quickly noted

Blood pressure instrumentation oscillometric tracings strain gauge capacitators, are all standard apparatus employed for *constant observation of cardiovascular function* during surgery The optimum chances of survival are thus much enhanced in modern hospital practice and the statistics are gratifying The experience gained in cardiac surgery with *therapeutic standstill of the heart* by *coronary instillation* of potassium salts and other drugs, has made cardiac arrest a commonplace occurrence and has stripped the syndrome of its former terrifying aspects With the possibility of stopping *and starting the heart at will* many new physiologic aspects of cardiac arrest have been learned

Subminimal Cardial Activity However the diagnosis may *not be so simplified, with minimal equipment and under the* dramatic circumstances of sudden cardiac collapse at the home or on a public thoroughfare the determination of standstill of the heart may be difficult and sometimes impossible *The absence of heart sounds failure to feel or see* the radial or any other arterial pulsation and the inability to obtain a blood pressure reading all *suggest* cardiac arrest but do not necessarily *prove* it Experimental studies in many *such cases seen especially in certain pre agonal conditions,* show considerable electrodynamic activity The ECG may present a more or less normal pattern but more often various grades of abnormal cardiac physiology are noted Such a *heart has not stopped even* though its functional capacity may have reached its lowest level

It is doubtful that measures necessary to reactivate the stopped heart serve any useful purpose here intracardial injections and open cardiac massage may do far more harm

than good In an individual dying from heart disease such manipulation can promise no improvement in the basic pathology It has already been emphasized that the mechanism of cardiac reactivation is chiefly based upon the physiologic factors asociated with myocardial hypoxia just so long as ventricular output is sufficient to maintain a minimal coronary flow lethal hypoxia will not occur Such minimal ventricular activity may be too feeble to produce the usual signs of peripheral circulation so that no pulse beat or blood pressure can be determined by the ordinary clinical methods of examination Ventricular contraction may be so reduced that no heart sounds are audible Patients may remain in this state of reduced cardiovascular function for a number of hours or longer in experimental hypothermic reduction of cardiac function in animals such a state may be continued for as long as several days with complete recovery Such a period of minimal function is not cardiac standstill but it may be a terminal phase of the failing heart Differential diagnosis is of the utmost importance here for the specific treatment of the two syndromes is entirely dissimilar

CLINICAL TESTS OF CARDIAC STANDSTILL

Clinical tests are defined in this context as those observations which may be made by the physician without the aid of special instrumental assistance and without the benefit of the factual data supplied by the operating room equipment previously mentioned With no more than his professional bag offers what procedures may the physician employ under emergency conditions? Unfortunately there is no general agreement either among cardiologists or cardiac surgeons concerning this very vital issue at a symposium called for

this purpose at the Valley Forge Heart Institute and Research Center in 1954 the following methods were considered to be within the scope of the internist's field of examination under the ordinary circumstances of such an emergency situation

1 *Intra Arterial Puncture* With a 50 mm needle and 1 10 cc glass syringe puncture is made into one of the larger available arteries the femoral in the inguinal region or the brachial high in the arm may be used The unattached needle is inserted into the artery first in cardiovascular collapse the procedure is simple to perform If blood appears in spurts regardless of how feeble this observation may be taken as evidence of ventricular contraction Experiments have shown that even when no blood pressure can be determined by conventional methods there may still be sufficient sub clinical arterial pressure to permit some volume of blood flow in the larger vessels If no spurting can be discovered and if there is a more or less free flow of blood through the needle the syringe should now be carefully attached and held in an upright position An upward movement of the plunger indicates intra arterial pressure pressure gradients as low as 2 to 5 mm Hg can be detected by this method

2 *Arterial Incision* If needle and syringe are not available a simple incision of a peripheral artery like the cubital may be quickly performed caution must be exercised to avoid complete severence of the artery In its more or less collapsed condition it may be cut across and may constitute a subsequent problem If spurting occurs some grade of hemody

namic integrity can be assumed simple oozing may occur in the early phase of cardiac arrest

3 *Pericardial Probing* A long intracardial injection needle from 10 to 15 cm in length may be inserted carefully into the 5th intercostal space near the sternum and slowly moved toward the heart The pericardium should not be pierced If there is ventricular contraction regardless of how limited there will be a characteristic feel given to the probing needle This feeling can sometimes be noted in ventricular fibrillation

None of these initial tests should require over 30 to 50 seconds to perform if actual standstill has occurred no further time should be wasted if one believes that arrest has taken place Prompt positive resuscitation must be started immediately together with artificial respiration if indicated

CARDIAC ARREST

	Complet Standstill	*Functional Standstill*
Physiologic mechanism	Sinus nodal pacemaker block	Ventricular fibrillation
Age incidence	Children and young *adults usually*	Middle and older age *groups*
Status of cardiovascular system prior to arrest	Usually normal	Usually abnormal coronary artery and rheumatic disease predominate
Chief initiating factors	Explosive vagal reflex mechanisms inhalation anesthesia trauma	Myocardial infarction congestive heart failure acute myocarditis toxic drug reactions physiologic shock accidental electrocution
Myocardial hypoxial reactions	3 stages usually clearly defined	3 stages usually very poorly defined
Reaction to extracardial procedures	Fair to excellent	Poor to none

Reaction to intra cardial procedures	Good to excellent	Poor to none
Reaction to artificial pacemaker	Good to excellent	Poor to none
Reaction to counter shock methods	Poor to none	Fair to none
Appearance of exposed heart	Complete standstill no motion	Vigorous to poor fibrillary movements of ventricles
Reaction to manual massage of heart	Good to excellent	Poor to none
Effective drugs	Adrenalin Isuprel atropine strychnine ether alcohol	Quinidine Pronestyl digitalis strophanthin Xylocaine potassium
Incidence of recurrence	Rare	Frequent
Post resuscitation complications	Less common	More common
Incidence of survival	Fair to excellent	Fair to none
Post resuscitation cardiac status	Usually return to normal	Increase in the basic cardiac pathology

Part II

THE GASTROINTESTINAL EMERGENCIES

SAMUEL WEISS M D F A C P F A C G D I M

XI ACUTE ABDOMINAL PAIN

The threshold for pain or discomfort varies distinctly in different individuals and at different times in the same person Some patients complain of severe epigastric pain one day and describe the same symptom as mere discomfort at another time Here one may explain the difference in the degree of intensity as due to fatigue or emotional strain It is also interesting to note that similar lesions may cause quite different sensations in two individuals One patient with a duodenal ulcer may describe the pain as agonizing and another may call it merely a slight gnawing sensation A patient who has gastritis may complain of hyperacidity another of hunger pain another of fullness Some patients are hyperesthetic and hypochondriac and their complaints have to be discounted—bearing in mind however the possibility that in due course of time they may develop a really serious symptom which should not be overlooked

INDIGESTION AS A SYMPTOM

The gallbladder patient usually complains of indigestion and this may include gastrodynia heartburn difficulty in swallowing sour stomach hyperacidity pyrosis water brash, acid regurgitation fullness bloating fermentation peristaltic unrest constipation diarrhea flatulence etc Almost always the use of the term indigestion by the patient is associated with unpleasant sensations referred to a fairly definite level of the abdomen Symptoms often do not indicate the seriousness of the underlying condition on the other hand intense gastric distress may be present without organic disease

LOCALIZED PAIN

There are exceptions to the rule that disturbances in a definite region of the digestive tract are referred to a specific level of the body This involves a consideration of the difference between indirect visceral pain and splanchnic visceral pain Ross in 1887 suggested that the unexplained sensations arising from a diseased viscus may be perceived as cutaneous or subcutaneous sensations his observations on the segmental distribution of pain arising from diseased viscera laid the foundation for the modern concept of *referred pain* Later Mackenzie and Head differed with Ross s belief that painful sensations are carried over the sympathetic nerve fibers, rather they are distributed over the course of the spinal nerves emerging from the cord and spread over their cutaneous ramifications Pain from a diseased organ is frequently noted at a point far distant and there are certain areas of cutaneous hyperesthesia following the same segmen

tal distribution In cases of esophageal disturbance pain is usually referred to the substernal area at a point corresponding to the level of the esophagus While stomach disturbances are most often referred to the middle or upper epigastric region duodenal pain is also referred to the same level Sensations arising from the small bowel are most frequently referred to the umbilical region and those from the large bowel to the midline in the hypogastrium

Splanchnic Pain Both Ross and Mackenzie asserted that in addition to referred somatic pain there is a deep heavy splanchnic pain felt in the region of the diseased organ This was substantiated by White Garrey and Atkins who showed in dogs that section of all the upper intercostal nerves does not diminish cardiac pain unless there is an interruption of sympathetic rami accomplished by section of the posterior spinal roots

Visceral Pain David Pollock and Stone in 1932 differentiated between the indirect referred type of visceral pain and direct pain carried by visceral nerves Pain caused in cats by distention of the gallbladder can be partly abolished by section of the intercostal nerves distal to the point of entrance of the sympathetic rami but complete freedom from pain is obtained only by resection of the right splanchnic nerve or by cutting the posterior root Soma Weiss and Davis whose experiments dealt with patients who had angina pectoris eliminated the referred pain by injecting novocaine over the cutaneous areas but some patients continued to have a deep sensation of discomfort These experimenters regarded their clinical work as corroborative of a viscerocutaneous *reflex*

since blockage of the normal stream of impulses from the skin eliminated any external response from the irritable focus in the spinal cord Zollinger however takes the view that this may also be accepted as supporting Morley s theory of peri*toneocutaneous reflexes as novocaine blocks impulses passing* through related nerve fibers between irritable parietal peritoneum and the overlying skin

Zollinger found that distention of the gallbladder by a small balloon was followed by deep epigastric discomfort absence of pain in the region of the gallbladder absence of vomiting absence of referred pain to the back or infrascapular region but was accompanied by respiratory difficulty particularly on inspiration When he distended the common duct epigastric distress was aggravated vomiting occurred there was no referred pain to the back or right quadrant and acute inspiratory distress was present He therefore suggested that involuntary vomiting would indicate the presence of a stone in the bile ducts

These considerations are of more than academic interest they are of importance in judging the patient s complaint of localized pain and especially in interpreting the areas of pain or tenderness found on physical examination of the abdomen Because this is at times difficult more and more reliance has been placed upon roentgenology laboratory tests and other special diagnostic measures such as cystoscopy and proctoscopy physical examination is unfortunately regarded as less important than heretofore Nevertheless it is a mistake to neglect the old methods of physical examinations namely inspection palpation percussion and auscultation of the abdomen The sensitive touch of the examining

hands must be taught to retain the ancient skill in all fairness to the patient as well as the physician

In connection with the foregoing discussion an outline is here presented of the segmental distribution of referred pain and tenderness in visceral disease —

SEGMENTAL DISTRIBUTION OF REFERRED PAIN AND TENDERNESS

Esophagus	Fifth sixth and eighth dorsal segments
Stomach	Third and fourth cervical and sixth seventh eighth and ninth dorsal segments Cardiac end from the sixth and seventh and the pyloric end from the ninth
Intestines	Down to the upper part of the rectum ninth tenth eleventh and twelfth dorsal segments
Rectum	Second third and fourth sacral segments
Liver and Gallbladder	Seventh eighth ninth and tenth dorsal segments and perhaps the sixth
Kidney and Ureter	Tenth eleventh and twelfth dorsal segments The nearer the lesion lies to the kidney the more is the pain and tenderness associated with the tenth dorsal segment The lower the lesion in the ureter the more does the first lumbar segment tend to appear

Bladder	Mucous membrane and neck of the bladder first second third and fourth sacral segments Over distention and ineffectual contraction eleventh and twelfth dorsal and first lumbar segments
Prostate	Tenth eleventh and twelfth dorsal first, second and third sacral and third lumbar segments

DIAGNOSTIC SIGNIFICANCE OF PAIN

By means of palpation the hand applies pressure for the purpose of obtaining more exact diagnostic information as to the location of existing pain or of discovering an unrevealed but suspected area of hyperalgesia Similar areas on both sides of the body should always be compared and zones of hyperesthesia should be outlined as accurately as possible Organs that touch the abdominal wall only in part —*e g* the stomach spleen liver intestine and kidney— should be percussed carefully for pain and tenderness The linea alba from xiphoid to symphysis should also be examined Sensibility to pressure should be tested over the region of the pylorus the gallbladder the three colonic flexures (hepatic splenic and sigmoid) the appendix the hernial openings and bladder ovary tubes uterus and kidneys

Location of Pain The physician must be careful in taking the history of the patient to question him closely regarding pain felt in any particular organ the patient s description should never be accepted without certain mental reservations A pain should not be accepted as localized in the stom

ach heart lungs etc but only in the region indicated or pointed out by the patient One should examine for areas of hyperalgesia of the skin or muscles for the visceromotor reflex (contracted muscles) and organic or functional symptoms of visceral disease When pain is the chief complaint the examiner should inquire particularly as to the earliest circumstances under which it was felt the place in which it was first noted and the areas into which it spread

A patient will often fail to describe a pain accurately he should be instructed to note these points the next occurrence In obscure cases it is often necessary to direct him to outline or mark upon his body the points of maximum intensity and the pain areas

Time of Occurrence Does the pain appear at any particular hour of the day or night, or after any physiological function (eating defecation urination etc)? Is it constant intermittent remittent or periodic? Is it worse at certain times of the year or during certain kinds of weather? Is it relieved by any special posture or action? The information gained as a result of these questions should be noted carefully it will have an important bearing upon the diagnosis

Intensity It is difficult to estimate or appreciate the intensity of pain given as a symptom It is greatly influenced by the temperament of the individual Neuropathic or neurasthenic individuals usually exaggerate the severity of pain Some patients complain bitterly of slight pain others pride them selves upon being stoical The facial expression sometimes gives valuable information Severe pain long continued affects nutrition and usually body weight The pulse and blood

pressure should be taken when pain is at its height and again in the intervals The size of the pupils when pain is complained of on pressure should be observed

Quality of Pain Is the pain of some peculiar character or quality as described by the patient namely lightning (tabes) gnawing (rheumatism) burning or tingling (neuritis) girdle (spinal) boring or pounding (aneurism or bone disease) leadcap (neurasthenia), brow recurring (malaria) throbbing (inflammation) shooting (neuralgia) stabbing or catching (pleuritic) griping or colicky (abdominal) cramping (muscular) nocturnal (syphilitic)?

Distribution Further questioning should lead one to decide whether or not the pain is limited to the cutaneous area of some peripheral nerve or whether it has the distribution of a spinal segment Is the pain preceded accompanied or followed by soreness hyperalgesia or hyperesthesia?

Influence of Position The existence of a position in which pain is aggravated is in favor of an organic lesion The presence of a painful position indicates the necessity for further investigation of the organ or new growth causing it The occurrence of a painful position points toward a localizing process

REFERRED PAIN

Episternal Pain This is produced by circulatory disease mediastinal disease or affections of the esophagus stomach or liver Pericarditis may give rise to intense persistent retrosternal pain Aortic aneurysm causes pain in the interscapu

lar region Radiation of headache into the interscapular space may be due to brain tumor or meningitis Neurotic tachycardia pseudo angina pectoris and true angina give rise to severe pain episternal or in the shoulder and arm Neurasthenia may also produce pain in the chest shoulder or arm this is relieved by motion not aggravated by it—a useful diagnostic point

Scapular Pain This may be caused by spinal disease muscles nerves pleura or organs below the diaphragm Pyloric stenosis may give rise to severe pain radiating into both scapular regions when the stomach is distended Gastric ulcer also causes scapular pain

Epigastric Pain This may be caused by muscular exertion due to persistent cough muscular hematoma the neuralgia of spondylitis locomotor ataxia (girdle pain) gastric crisis acute gastritis gastric ulcer cholelithiasis cancer of the liver epigastric hernia gastralgia lead colic pyloric stenosis splenic tumor ecchinococcus disease lower esophageal stenosis intermittent dilatation of the aorta abdominal hepatic congestion or abscess or paraesophageal hernia (hiatus hernia) Again it may be caused by diaphragmatic pleurisy subphrenic abscess thrombosis of the superior mesenteric artery acute inflammation cysts or hemorrhage into the pancreas

Other symptoms such as nausea anorexia slight loss of weight occasional vomiting may be caused by tumor of the brain psychoneuroses and general disturbances of the vegetative nervous system Cardiac and kidney diseases bladder and prostatic affections a distantly located carcinoma pregnancy infectious diseases and intoxications alcoholic gas

pressure should be taken when pain is at its height and again in the intervals The size of the pupils when pain is complained of on pressure should be observed

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REFERRED PAIN

Episternal Pain This is produced by circulatory disease mediastinal disease or affections of the esophagus stomach or liver Pericarditis may give rise to intense persistent retrosternal pain Aortic aneurysm causes pain in the interscapu

plete obstruction If rectal examination demonstrates a mass of hard packed feces attempt should be made to remove the feces with the fingers In women posterior and downward pressure through the vagina may cause relaxation of the rectal sphincter to permit passage of the hard mass Morphine or other opiates should not be used because they prevent relaxation of the sphincter however when not contraindicated Demerol or rapid acting barbiturates may be employed Where failure to remove the hard mass occurs *hospitalization and light anesthesia may be necessary* on the other hand warm oil enemas or an enema with one or two tablespoons of peroxide of hydrogen in a glass of warm water may be effective

HEMORRHOIDS

Hemorrhoids thrombosed or not thrombosed may also constitute a gastrointestinal emergency A patient with prolapsed or protruding hemorrhoids may have severe lower abdominal pain simulating appendiceal or bladder stone In failing to examine the anal area the examiner may miss the cause of the pain Reduction of prolapsed or protruding masses is best accomplished by a well lubricated gloved finger If this cannot be done without additional pain or trauma because of possible strangulation injection of hyaluronidase 2 cc in 0 5 per cent procaine after a period of five or ten minutes will permit replacement of the entire mass Warm baths and the insertion of suppositories containing Benzocaine with other soothing agents may some times prevent recurrence Care of the bowels by diet and medication internally to soften the feces are also indicated

When hemorrhoids are thrombosed and very painful in

jection of 1 per cent procaine or Xylocaine and evacuation of clots through a small incision may be necessary I have found that after injecting procaine or Xylocaine and leaving the needle in situ for several minutes withdrawal of the piston of the syringe slowly will in many instances evacuate blood and cause collapse of the thrombosed hemorrhoids this should be followed by the usual treatment and diet

*When this is not feasible witchhasel diluted with water ap*plies to the thrombosed hemorrhoid together with a light ice cap will bring relief in many patients Care should be taken that hypothermia is not constant but intermittent

HERNIA

Here as in other abdominal manifestations the physician must not neglect to examine the patient s lower abdomen small hernias even when not strangulated may give pain in the lower abdomen radiating to the lower back and even down the thigh These hernias may be either inguinal femoral or ventral They may simulate stone in the bladder appendicitis or stone in the lower ureter

Where reduction is possible it may best be accomplished by the application of warm moist cloths placing the patient in the semi Trendelenburg position or spraying Ethyl chloride from a distance of 10 12 inches being careful not to freeze the skin too much After reduction a proper supporter or temporary truss may help to keep the hernia within bounds *Where negative results are obtained* after a reasonable time the patient should be hospitalized for further attempts to reduce the hernia under light anesthesia or if indicated for immediate operation

HICCUPS

Hiccups may also become a gastrointestinal emergency when overdistention of the stomach by food or swallowed air occurs Emergency relief may be obtained by gentle pressure over the stomach area or by administering of a half teaspoonful of bicarbonate of soda in half a glass of water with or without lemon juice

Various other remedies may be tried such as application of ice to the back of the neck swallowing small pieces of ice pulling on the tongue rebreathing into a paper bag gastric lavage Compazine by hypodermic inhalation of 5 or 10% CO in oxygen if available is of real value in many cases

A Final Word In conclusion it is essential to stress the importance of abdominal pain from a diagnostic viewpoint The differential diagnosis of abdominal diseases may be difficult for it is not always possible to correlate any one symptom with a given organ There are many reasons for this difficulty In the abdominal cavity a number of organs lie in close proximity with one another and influence each other functionally These organs are also connected with each other by the visceral nervous system so that radiation of pain occurs and the primary site of the disease be missed Many of the abdominal organs radiate similar sensations of pain which often are of a spastic type these stimuli may originate in the stomach the intestines the bile ducts or the urinary tract (dyskinesia) If there are multiple displacements of viscera topography will be of little or no value in determining the exact organ causing the pain Although

most of the hepatic and splenic conditions lend themselves readily to recognition because of their location a floating or displaced kidney liver or spleen may present diagnostic problems The same is true perhaps in a greater extent in various displacements and ectasias of the stomach In organic gastrectasia one may find the pylorus laterally on the right side especially in carcinoma of the pylorus The latter may also lie deep in the hypogastrium when displaced by adhesions or specific functional conditions Foerster has called attention to a perpendicular or transverse position of the stomach depending upon the contractions of the longitudinal or transverse musculature

Again difficulty in diagnosis may be due to an inaccurate or incomplete history when the patient has withheld important data In elderly individuals who are less sensitive to pain stimuli than younger persons the physician may be misled and be unable to evaluate accurately certain symptoms Puzzling as a case may be however if the physician finds the topography of the viscera not absolutely normal the location of the colicky pain may still be of help in diagnosis

The clinician must ever be on guard remembering that mild general symptoms referable to the gastrointestinal tract may be manifestations of disturbance in organs entirely unrelated to the abdomen

XII UNCONTROLLED NAUSEA AND VOMITING

The physician summoned to treat a patient with nausea and vomiting should first differentiate between *regurgitation* and *vomiting* When an individual regurgitates it is ordinarily without effort there is neither nausea nor retching Food or fluid comes back into the mouth without emptying the stomach completely According to Alvarez regurgitation begins usually during a meal or soon after it and continues coming up in mouthfuls In some instances there is a familial background (psychotic forebears)

In many of these individuals regurgitation may increase after an emotional upset or if the patient gets tired or hysterical Loss of weight dehydration and cachectic symptoms may appear in girls or mature women who develop nervous anorexia Instances may occur where an individual is allergic to certain foods the latter may be remedied when the known allergen is avoided

The important differential diagnostic point is that the patient is only bringing up mouthfuls of fluid or food and not emptying the stomach completely by actual vomiting

The treatment is suggestive preventing emotional upsets avoiding food to which the patient is allergic regulating bowel movements if constipated Medical treatment may be

employed with special attention to the tranquilizers Thorazine or Compazine oral hypodermic or rectal suppositories Perphenazine (Trilafon) Bonamine or similar preparations may be helpful

Examination of Vomitus The physician when examining the vomitus may note absence of food this speaks against pyloric spasm or obstruction The presence of blood or bile does not always spell ulcer cancer or liver disease Blood may be present from the strain of vomiting or due to a tear at the esophago cardiac junction Bile may be due to reverse peristalsis especially characteristic in migrainous headache Projectile vomiting is characteristic of brain tumor and absence of nausea is a diagnostic feature When nausea and vomiting with dizziness occurs in an elderly or hypertensive individual and not associated with ear involvement thrombosis or spasm of the smaller arteries of the brain should be kept in mind

TREATMENT

The treatment in vomiting depends upon the underlying cause after a definite diagnosis has been established When vomiting is persistent the oral administration of drugs must be avoided Attempts should be made to quiet the gastrointestinal tract by hypodermic or rectal medication When vomiting is due to overeating emptying of the stomach by emesis or lavage is indicated concentrated saline solution or mustard one tablespoon to a cup of warm water gastric lavage through a stomach tube using warm saline or bicarbonate solution will often relieve further distress Thorazine or Perphenazine hypodermically or a rectal suppository

apomorphine hydrochloride (gr 1/10) subcutaneously or intramuscularly may be employed When the patient vomits profusely is comatose or greatly depressed do not use emetics! Intramuscular injection of Gynergen if given early will relieve vomiting caused by migraine Do not use morphine or Demerol its use may increase vomiting Suppositories containing Nembutal Butisol or sodium amytal are useful A low enema may often be helpful

XIII THE ACUTE ESOPHAGEAL SYNDROMES

Dysphagia or difficulty in swallowing is the most common symptom in the acute disturbances of the esophageal tract from a clinical point of view, the esophageal tract can be defined as extending from the mouth to the cardia of the stomach The muscles of deglutition are functionally competent below the level of their anatomic area and there is considerable *overlapping of central and sympathetic enervation* hypoglossal fibres may extend as low as the middle third of the esophagus Subjective symptoms of esophageal dysfunction may thus present a syndrome of varying complaints which may have little or no correlation with the actual pathology present in any given case The most frequent lesions are inflammatory processes and obstructions due to benign or malignant conditions Neurologic manifestations of central origin usually involve the upper part of the esophagus cardiospasm of sympathetic origin is ordinarily confined to the middle and lower portions

Painful swallowing may originate in the mouth pharynx or the esophagus *Spasm of the muscles may be due to the* meeting of two peristaltic waves acute distention and swallowing corrosive substances A piece of chicken bone swallowed accidentally may pierce the esophagus causing medi

phy shows bulging of the esophagus especially at the lower end while esophagoscopy especially in advanced cases reveals a dry reddened mucosa with areas of erosions and ulcerations

TREATMENT

Treatment consists of avoidance of all highly seasoned foods extremes of hot and cold drinks spices raw fruits, vegetables and roughage Prescribing of antispasmodic and topical anesthetic drugs demulcents bismuth subcarbonate and pureing all foods may give relief in the early stages Also in the early stages the introduction of the esophagoscope or a large stomach tube and Hurst s mercury filled esophageal rubber tube may give some or complete relief However where degeneration of the neuromuscular mechanism is present these treatments will be useless and sometimes dangerous

More advanced achalasia should be treated by dilatation with hydrostatic dilators Failure to improve and where dilatation and sacculation are marked surgery is preferable to repeated dilatations

Another condition which may cause obstruction of the esophagus is a traction diverticulum Simple diverticula may not cause acute or chronic obstructive symptoms however when a diverticulum is the result of scar contractures from tuberculous mediastinal lymph nodes and the inflammatory process involves the esophageal wall there may be dysphagia, regurgitation and pain

Sensory and motor paralysis may cause dysphagia Diagnosis is established by direct examination of the pharynx

which may reveal a flaccid and toneless pharynx cricopharyngeus muscle and upper esophagus

ESOPHAGEAL HEMORRHAGE

Esophageal hemorrhage may be caused by varices of the esophagus secondary to cirrhosis of the liver Banti s disease, and splenic anemia These varices are dilated tortuous veins in the submucosa of the lower esophagus at times extending well up into the esophagus and down into the cardiac end of the stomach Esophageal varices should be suspected in the presence of hematemesis and melena The finding of an enlarged spleen in a patient with gastrointestinal hemorrhage should make the physician suspicious of esophageal varices

Roentgenography with barium reveals the presence of these varices in the esophagus When the varices are not too prominent or advanced negative findings both with the x rays and the esophagoscope is not uncommon

TREATMENT

When hemorrhage occurs immediate attempt should be made to arrest the bleeding One of the most efficient methods is to introduce the Sanstaken Blakemore nasogastric tube inflation will cause local pressure and tamponade sufficient to collapse the veins in the coronary esophageal collateral circuit Regurgitation reflexes must be abolished by deep sedation and even by anesthesia in certain cases Transfusions Vitamin K and C by hypodermic administration are additional safeguards Some patients with severe liver failure may not respond Injection of sclerosing solutions into the varices through the esophagoscope topical thrombin appli

cation through the esophagoscope, followed by 5 cc of a 1 10 solution Thrombin Topical at 30 minute intervals for a total of four doses may be necessary The solution seeps down around the tamponade and provides additional hemo stasis where oozing continues Surgical exposure and ligation of the varices within the esophagus with or without simulta neous injections of sclerosing solutions into the varices has been used Postcaval venous shunt or splenorenal venous shunt procedures may be life saving Where the damaged liver cannot maintain a safe prothrombin level surgery may fail to save the patient

PSYCHOGENIC DYSPHAGIA

Acute esophageal syndromes are not uncommon in psychiatric and highly emotional individuals the etiological background may be difficult to determine when actual cardiospasm is present The tranquilizing drugs may have a valuable thera peutic role here in addition to the preparations employed in the treatment of gastric regurgitation The heartburn which originates at the lower end of the esophagus may be due to a highly acidogenic seepage from the cardia which is not infrequent under emotional stress even in otherwise nor mal individuals

Injuries to the esophageal mucosa following the ingestion of small foreign bodies are also common in mentally dis turbed patients Foreign bodies may cause perforation at any point in the esophageal tract massive infection of the mediastinum may produce few if any early symptoms in such cases X ray will reveal the type and number of opaque foreign bodies but the non opaque like wooden or plastic

buttons pegs and certain toilet articles may require special methods of visualization

Repeated Patterns Psychotic patients have a tendency to repeat the dysphagic pattern when the history reveals a number of episodes of difficult swallowing foreign body ingestion should be suspected

ACCIDENTAL ESOPHAGEAL OBSTRUCTION

Partial or complete esophageal obstruction following the accidental or unintentional swallowing of a large bolus of food or other material may become an acute emergency problem If the mass is caught at the upper end of the esophagus at the level or just below the larynx there may be a prompt associated spasm of the upper airway with tracheal and main bronchi involvement This syndrome must be differentiated from the patient who "chokes to death" from a piece of food in this instance the foreign body has been inspired into the trachea and blocks respiration This is an emergency of high priority if the mass can not be removed by the fingers or by available instruments an immediate tracheotomy is indicated In the reflex respiratory spasm due to acute esophageal obstruction tracheotomy is rarely necessary any of the spasmolytic drugs may be used as a temporary measure Removal of the food bolus may be a simple procedure after the spastic reaction has disappeared Such masses should be withdrawn upward from the obstructive area it may be possible to fragment the bolus so that it may be recovered in several smaller pieces No attempt should ever be made to force the mass downward

ASPIRATION PNEUMONIA

During removal of esophageal foreign bodies aspiration of small bits of the mass is the rule the stricken individual in his frantic efforts to breathe may inspire infective material from the mouth Wide spectrum antibiotics must be promptly employed the chief organism is usually a resistant staphylococcus All emergency hospitals are reporting a serious increase in the mortality from this type of aspiration pneumonia

XIV ACUTE ASPECTS OF HIATUS HERNIA

Hiatus hernia when symptomatic may stimulate cardiac pulmonary or gastrointestinal disease the syndrome has been referred to in the chapters concerned with heart pain and certain diseases of the lung and pleura It is considered here in detail since the condition primarily involves the gastrointestinal tract

HERNIAS OF THE DIAPHRAGM

These are classified into three main groups congenital acquired and traumatic The congenital type due to embryologic deficiency are usually without an enclosing sac The most common sites of occurrence are through the foramen of Bochdalek through the esophageal opening through the foramen of Morgagni and through the gap left by absence of left half of the diaphragm Hernias acquired after birth are found through a point of embryologic fusion of the diaphragm at sites named under congenital types and through the esophageal hiatus in the latter type the hernia has an enclosing sac Traumatic diaphragmatic hernias may be divided into those due to direct injury to the diaphragm which may occur at any point these include points of embryologic fusion most commonly in the dome and posterior

half of the left side of the diaphragm and usually is the result of a severe crushing injury When the hernia is through the esophageal opening there is a sac but when the hernia is through the leaf of the diaphragm there is no sac Hernia due to direct injury to the diaphragm which may occur at any point is usually the result of a penetrating wound from a gunshot or a knife or from rupture of a subdiaphragmatic abscess or empyema here ordinarily there is no sac

The clinical recognition of a diaphragmatic hernia is often difficult because the symptoms produced often simulate those of other diseases of the abdomen and thorax of which the most common are cholecystitis gastric ulcer achalasia angina pectoris intestinal obstruction and unexplained secondary anemia

Symptoms Two main groups of symptoms may occur in diaphragmatic hernia first where only the stomach is involved and in which the hernia is usually of the esophageal type in the second group multiple abdominal viscera are involved and in most instances are caused by trauma As more of the stomach becomes involved in the hernia the attacks become more pronounced the pain is projected *straight through to the back and to the lower left side of the thorax it is more marked to the left of the spinal column* and may appear between the shoulder blades The pain may be agonizing there is difficulty in belching of gas and vomiting because of spasm of the diaphragm and reflex cardiospasm Hourglass deformity of the stomach occurs preventing emptying of the herniated portion and causing increased *intragastric pressure*

Spasm of the diaphragm may cause referred phrenic nerve pain in the left shoulder which at times involves the entire

left arm When cardiac embarrassment with palpitation tachycardia and dyspnea occur differential diagnosis may be difficult Lying down causes the symptoms to increase and in some cases the patient prefers to sit up in order to breathe The attacks may last for a few minutes to several hours Vomiting usually relieves the attack episodes often recur immediately after food is taken

Often patients are relieved for a time when placed on ulcer regimen because of the restricted amount of food intake antispasmodic and antacid medication

HEMORRHAGE

This is not a usual occurrence of hiatus hernia when it develops it may be caused by erosion of the mucosa an ulcer or severe incarceration of the hernia with fixation of the stomach in the thorax

TRAUMATIC HERNIA

In traumatic hernia where the stomach alone is involved the symptoms are practically the same as in esophageal hernia In most cases of traumatic hernia the colon is involved and there are added symptoms caused by interference with its function Obstinate constipation with accumulation of large quantities of gas in the colon or periodic attacks of complete obstruction severe gastric hemorrhage and marked respiratory symptoms caused by the large amount of viscera in the thorax produce pressure on the lungs and pericardium

In cases where the diagnosis has established that the symptoms are caused by esophageal hernia we have been successful in many instances in relieving the patient with 1/100 grain of nitroglycerin sublingually or a rectal suppository

containing 3½ to 7 grains Aminophylline 1 grain of Papaverine or Seconal Morphine or Demerol should be avoided as it may increase nausea and vomiting

Surgery Surgery may be necessary when medical procedures fail although the surgical approach to the herniated area may present many technical difficulties Indications for surgery may depend upon a number of complicating factors where there is more or less cardiac disability as in coronary or advanced valvular disease the secondary effects of surgical interference may enhance the pre existing heart disease There are less contraindications in pulmonary conditions Following successful surgery there may develop a postoperative syndrome similar to that described after gallbladder removal in which the symptoms imitate within certain limitations those which existed before operation Successful reduction of the herniated viscus may not necessarily be followed by complete relief of symptoms in this condition the entire problem of hiatus hernial symptomatology remains to be satisfactorily explained since many demonstrated hernias of the diaphragm produce no symptoms throughout the entire life time of certain individuals regardless of the size shape and position of the lesion These patients stand in direct contrast to those who suffer excruciating episodes of pain from minor hernial pathology

Prognosis Conservative and symptomatic treatment of the acute episodes offers the best long range outlook in the management of these difficult cases where complete obstruction occurs surgery is of course, mandatory In other cases the patient should be told that no promise of complete relief

can be given regardless of the type of surgery performed From a more optimistic point of view it should be pointed out that there may be long spontaneous remissions in the syndrome many patients have a history of one to 5 or 6 attacks over a short period and then enjoy several years of symptom free relief

XV ULCER PERFORATION

Perforation is the most serious complication of ulcer and it is estimated to occur in about 10 15 per cent of all cases of chronic gastric and duodenal ulcer It is more frequent in duodenal ulcer than in gastric ulcer both types occur less frequently in women

In acute perforation a sudden rupture of the base of a gastric or duodenal ulcer takes place contents of the stomach or duodenum may escape into the general peritoneal cavity

Symptoms When perforation takes place the patient experiences agonizing abdominal pain and makes every effort to splint the abdomen and avoid undue movement Respirations are costal and shallow and beads of sweat appear on the forehead There are no immediate evidences of peripheral vascular collapse the pulse may be normal or slightly elevated blood pressure is not altered significantly temperature remains normal or subnormal The abdomen is board like and tender over the site of the ulcer Liver dullness may be obliterated in some cases and auscultation of the abdomen may show absence of peristaltic sounds initially but these may return later

Types of Perforation Three types of perforation are described

Acute The ulcer perforates and the general peritoneal cavity becomes flooded with gastric or duodenal contents *Subacute* In subacute perforation only a circumscribed area of the peritoneal cavity becomes contaminated by the leakage *Chronic* Here the ulcer has penetrated a neighboring viscus such as the pancreas Sometimes as the result of a slow leak a localized abscess may develop in the vicinity of the ulcer

Peptic Ulcer Ninety five per cent of peptic ulcers which perforate into the general peritoneal cavity are situated on the anterior or anteroposterior wall of the stomach or duodenum Posterior *ulcers which often penetrate deeply into the substance of the pancreas can only perforate* when the ulcer spreads and burrows its way toward the superior or inferior surface of the duodenum Multiple perforations occurring simultaneously have been described but they are very rare in our experience

DIAGNOSIS OF PERFORATION

In diagnosing perforation of an ulcer the physician must recognize the chief symptoms changes in the pulse rate temperature lowering of the blood pressure marked leukocytosis and the classic signs of peritonitis with shock predominate Increased amylase in the blood or urine may be present when the pancreas is also involved To obviate errors in diagnosis the examiner must differentiate perforation from biliary or renal colic intestinal obstruction acute appendicitis acute pancreatitis perforation of the gallbladder or the hepatic or splenic flexures of the colon Sometimes tender

ness may shift to the right lower quadrant or it may remain generalized depending on the spread of the gastric contents along the abdominal gutters An x ray film of the abdomen will show the presence of air under the diaphragm however the absence of air under the diaphragm does not rule out perforation

Subacute and Chronic Perforation In subacute or chronic perforation a less dramatic picture is seen In some of these cases a subphrenic abscess may form Early spontaneous closure may occur in minor perforations the symptoms become less and less and may disappear within 10 hours

Prognosis The prognosis in perforations depend upon early diagnosis and operation Usually best results are obtained when the patient is operated on within the six hour period after perforation had taken place

Where spontaneous closure occurs watchful waiting the use of Demerol and Wangensteen tube and intravenous medication may tide the patient over the critical period

XVI GASTRIC HEMORRHAGE

Clinical diagnosis of the exact site of hemorrhage must first differentiate between gastric and esophageal hemorrhage hemorrhage from the lungs and other organs The symptoms of massive hemorrhage are faintness weakness dizziness and *sweating followed by vomiting of blood or tarry stools When the hemorrhage is severe syncope collapse with low* ering of the blood pressure weak and rapid pulse and excessive thirst develop The blood count and hemoglobin in the early stages may show little or no change later marked diminution of these elements are found The BUN is mod erately elevated because of absorption of blood from the intestine and from changes in kidney function secondary to the lowered blood pressure

Site of Bleeding In differentiating gastric from duodenal bleeding it should be remembered that hematemesis alone occurs more frequently in gastric ulcer than in duodenal ulcer and melena is more frequent in a duodenal or in a marginal than in gastric ulcer Melena without hematemesis may occur with esophageal varices The black and shiny stools seen after gastric or duodenal hemorrhage are caused by acid hematin from hemoglobin due to the action of acid

At times when blood passes rapidly through the stomach the color may be dark red and here the question arises from whence the blood originated *In general blood from the* intestine and colon is red and not thoroughly mixed with the stool when the blood originates in the duodenum or stomach it is tarry or black Confusion may also arise when the vomited blood is coffee ground or red Most authors believe *that coffee ground vomitus is characteristic of gastric carcinoma* some found red blood vomited in cancer of the stomach while the coffee ground type has been vomited in some ulcer cases Another source of error lies in the presence of hematemesis and hemoptysis when this differentiation is questionable *hematemesis, as a rule, is mixed with food and/or gastric juice, hemoptysis is bright red and frothy* Further differentiation may have to be made when blood from the nose pharynx or the lung is swallowed and then vomited Examination of the sputum if it is blood streaked and x ray *of the chest is indicated* especially *when there is a* history of cough

X ray and Gastroscopy Whether or not it is advisable to x ray the stomach or gastroscope the patient during the early stages of hemorrhage is *still a debatable question* When acute symptoms and shock are under control Hampton Schatski and others advocate using a thin mixture of barium while the patient is lying horizontally on the x ray table and avoiding manipulation over the stomach area When to gastroscope the patient after *gastric hemorrhage has also produced* conflicting opinions Some authors advocate early gastroscopy while others prefer to wait at least 10 days after

subsidence of the bleeding When the operation is to be performed early gastroscopy may aid in the preoperative diagnosis

Eye Changes Many patients complain of eye disturbances several days after bleeding in a routine ophthalmoloscopic examination of the eye grounds in many patients the white disc has been found edematous As the blood picture improves eye symptoms regress Several reports in the medical literature relative to eye changes noted amaurosis following hemorrhage usually more after repeated small hemorrhages than after a single large one According to these authors optic nerve atrophy was found which began immediately or was delayed more than 18 days after the episode

Temperature The presence of fever in some patients after the hemorrhage still needs clarification as well as the use of the duodenal tube to determine the continuation of bleeding or for lavage of the bleeding stomach

TREATMENT

The treatment of hematemesis and melena should be immediate Complete physical and mental rest should be instituted over zealous relatives and friends should be restricted from conversing or disturbing the patient Sitting up or effort at stool may bring on syncope and blackout Typing to determine the Rh and blood group should be done promptly matched blood should be available and given intravenously as soon as possible To prevent restlessness and fretting phenobarbital sodium injection N F one or two cc Dilau

did gr 1/48 repeated at 4 to 6 hour intervals hypodermically is less toxic and may prevent the vomiting which is seen in morphine sedation

Intravenous Medication When blood or plasma is not immediately available a 5 per cent glucose in saline solution given slowly intravenously will elevate blood pressure and reduce the pulse rate When nausea and bleeding continues or the patient has not recovered from shock oral feeding is to be avoided on the other hand if the patient is alert and not nauseous feeding can be started by giving milk and cream or equal parts of cream and gelatine

Andresen s Mixture

Jello 1 ounce or gelatine 3/4 ounces
Lactose 3 ounces
Juice of 2 oranges
Water 1 quart
Powdered sugar 2 tablespoonfuls

Feed 3 ounces every 2 hours while awake or

Gelatine 1 ounce
Sugar 2 ounces
Cream (20%) 3 ounces
Milk 27 ounces

Feed 4 ounces every 1 or 2 hours 1st and 2nd day

Delafield s Mixture

Cream 4 ounces
Milk 4 ounces

Vichy 4 ounces
Soda Bicarbonate 20 grains (Sodium Citrate is preferable as this does not form CO)
Cerium Oxalate 10 grains

Feed 1 to 4 teaspoonfuls every 1 or 2 hours first and second day

Vitamin K and Vitamin C may be given parenterally The flavonoids and toluidine blue may offer some help in controlling bleeding Laxatives and enemas are to be avoided at least for four or five days after bleeding has stopped For the secondary anemia iron preparations with or without vitamin combinations will be indicated after the stools return to normal color Surgery is indicated when repeated transfusions fail or recurrence of severe bleeding occurs When there is no danger of syncope or shock the patient may sit up in bed and dangle his feet or sit in an easy chair at the bedside At least two weeks should elapse before the patient is discharged from the hospital or allowed to go outdoors if at home

DIFFERENTIAL DIAGNOSIS OF EXTRA ABDOMINAL AND INTRA ABDOMINAL HEMORRHAGE

EXTRA ABDOMINAL	INTRA ABDOMINAL
Pulmonary hemorrhage—blood may be swallowed and later vomited It may resemble coffee grounds Important to differentiate although it may	Arteriosclerotic vessel gastric aneurism of hepatic artery appendicitis (chronic)—bleeding due to secondary congestion of stomach Bantis syn

be difficult early Auscultation of anterior chest and apices may show rales due to blood in small bronchi Blood from lungs is usually red and frothy and does not react to Congo red or Toepfers paper Less hypovemia and collapse than in intra abdominal hemorrhage The skin is less cold and clammy Morphine or Demerol relieves the restlessness more than in gastric hemorrhage A moderate elevation of temperature may occur

Blood dyscrasias—aplastic anemia hemophilia Hodgkins disease splenic anemia polycythemia thrombocytopenia contracted kidney carcinoma of the esophagus emboli (septic) nosebleeds systemic diseases acute endocarditis bacteremia cholera diphtheria malaria *measles scarlet fever variola* yellow fever various toxemias telangiectasis (familial) small varices are found on the front back or undersurface of the tongue *vicarious menstruation* drome benign tumors of stomach and duodenum bleeding by diapedesis bleeding gall bladder (may show signs of anemia blood in gastric contents or stool) cancer of stomach cancer of duodenum carcinomatous erosion of Vaters papilla cholecystoduodenal fistula cirrhosis of the liver diaphragmatic hernia (hematemesis or melena) diverticulum of sigmoid epithelioma (differential diagnosis—operation or autopsy) gumma (Wassermann luetic history ulcer or cancer and syphilis may be present) gastritis duodenitis mucosal erosions herniation of the gastric mucosa into the duodenum hiatus hernia lymphatic leukemia (chronic) Meckels diverticulum jaundice neurofibroma in transverse mesocolon *polyp (multiple or single)* sarcoma (gastric intestinal) difficult to differentiate between these and duodenal ulcer trauma—a blow or crushing injury *to the* abdomen may traumatize the wall of the viscus causing bleeding Gunshot or knife wounds Intubation of the

stomach Swallowed glass or metal bezoar tuberculosis of stomach and duodenum varicosities of the esophagus and stomach vascularity of stomach and duodenum (excessive) subserosal leiomyoma

Erosions or lacerations in the long axis of the stomach and esophagus should be suspected when hematemesis or a hemothorax follows a period of violent retching and vomiting from any cause

DIFFERENTIAL DIAGNOSIS OF HEMORRHAGE FROM STOMACH AND FROM THROAT AND LUNGS

HEMORRHAGE FROM THE STOMACH

History of organic disease of the stomach or of cardiac embarrassment with tricuspid regurgitation

Previous attacks of eructations of gas liquids and food that excited substernal burning

Study of regurgitated fluid shows it to contain either hydrochloric or lactic acid particles of food may be present

HEMORRHAGE FROM THE THROAT AND LUNGS

History of pulmonary disease with cough and expectoration

Paroxysmal attacks of coughing which develop upon rising in the morning

Material alkaline in reaction may contain tubercle bacilli and shreds of elastic tissue

Substernal oppression and discomfort An annoying sensation in the throat and a saline

Epigastric uneasiness precedes hemorrhage from the stomach nausea faintness and an acid taste are also experienced	taste precedes hemorrhage from the lungs
Blood and gastric contents are ejected by vomiting	Blood expelled first by coughing but vomiting is common later
Microscopically bloody fluid may contain clots of a dark brownish color ("coffee ground" vomitus) In profuse hemorrhage bloody fluid shows no clotting and contains particles of food	Fluid bright red its surface is beaded with froth Small blood clots seen occasionally
No external evidence of hemorrhage between attacks of vomiting	Patient continues to expectorate blood streaked sputum for hours or days after the hemorrhage

Normal hemostasis depends on the integrity of three basic elements the vascular wall the platelet and the coagulation of blood The presence of a bleeding tendency is usually revealed in the patient s history reports of spontaneous bleeding will be found in severe cases while in those that are less severe bleeding will be noted only after major injury or surgical and dental procedures Laboratory procedures are needed however both to confirm the existence of the tendency and to indicate its probable cause whether vascular defect platelet deficiency or coagulation abnormality The tourniquet test one of the simplest and most valuable of the screening procedures supplies information about both platelet sufficiency and vascular integrity

Most hemorrhagic disorders can be controlled if the remedies now available are properly used Patients with occult

bleeding tendencies that is those that do not bleed spontaneously but who will bleed severely during surgical procedures or after trauma usually have either pseudo hemophilia thrombocytoasthenia or the hemophilia like diseases They too can usually be detected by adequate preoperative screening tests More specialized problems are presented by the hemorrhagic syndromes directly due to surgical procedures and appearing in patients whose hemostatic mechanism was normal before operation One of these thrombocytopenia caused by multiple replacement transfusions is due to platelet deficiency induced by an abundant loss of blood and its replacement—poor bank blood Bleeding of this type can easily be controlled by the administration of platelet rich blood or of prepared platelets

fore removing the duodenal tube aluminum gel injected through the tube to bathe the inflamed area should be made

DUODENAL DIVERTICULA

Duodenal diverticula may occur within the wall of the duodenum these may be congenital or acquired The acquired diverticulum may be the result of an ulcer causing adhesions or scars around the lesion which contract or pull upon the wall The symptoms are either mild or severe pain or distress in the epigastrium nausea belching and vomiting The characteristic x ray findings in a diverticulum is the retention of barium for many hours or days

The treatment depends upon the severity of the symptoms Mild cases are treated with a bland diet avoidance of overeating and medication to minimize irritability or spasm of the duodenum When the diverticulum becomes infected diverticulitis occurs this may require rest in bed for a week or longer with restricted diet and medication Severe cases or rupture require immediate surgery

DUODENAL STASIS

Duodenal stasis in reality is not a disease entity but patients with visceroptosis often complain of bizarre symptoms pain nausea vomiting loss of weight migraine at times anemia and dehydration This syndrome may also be caused by adhesions about the duodenum resulting from previous inflammation of the organs in the immediate neighborhood or following surgery In many of these cases food and bile regurgitate into the stomach and the patient has many symptoms *Cysts of the duodenum are rare their presence may* cause clinical syndromes of intermittent duodenal obstruc

tion with similar roentgenologic and gross pathologic findings There are no data available concerning the age and sex distribution of cysts of Brunner s glands Various studies including x ray examination will furnish corroborative evidence of the presence or absence of malignancy

If not malignant prolonged rest diet and medication plus a good fitting abdominal support may offer favorable response in relieving the distress

JEJUNAL ULCER

On rare occasions an ulcer may manifest itself at some part in the intestine other than the duodenum The jejunum is the most likely location especially after gastroenterostomy or subtotal gastrectomy For jejunal or marginal ulcer the treatment is rest in bed restricted diet antacids duodenal feeding Recurrent or intractable cases require surgery In previous years when typhoid fever was prevalent ulcer of the ileum was frequent

INTESTINAL DYSPEPSIA

Intestinal dyspepsia is characterized by colicky or tearing types of pain it is spoken of as enterospasm whereas excessive distention of the lower abdomen is caused by flatulence In most cases it is due to rapid emptying of the stomach or small intestine resulting in food which has undergone incomplete digestion before reaching the large intestine The stool examination reveals a light foamy bulky sour smelling excrement acid in reaction with evidence under the microscope of undigested starch cells when the stool reveals a darker than usual color a not too bulky mass but a foul smelling putrefactive excrement usually neutral or alkaline

in reaction the condition is referred to as *putrefactive intestinal indigestion* At times both conditions may prevail

The symptoms vary at times diarrhea at other times constipation is present Most of these patients have a sallow, yellowish skin are generally irritable in nature have indigestion loss of appetite, thirst and a coated tongue Headache nausea vomiting anemia and nervousness are probably the results of autointoxication

Treatment is directed toward correcting or alleviating the symptoms and controlling the diet Medication and other measures are indicated according to the type of putrefaction present Medication to diminish or destroy bacterial decomposition and intestinal antiseptics orally or through the duodenal tube may be employed Operation is not indicated

INTESTINAL STASIS

This may be due to adhesion bands kinking of the bowel at various points disturbances in the reserve supply of the intestine at certain sites prolapse of the bowel or ptosis of the intestines usually the result of prolonged constipation The symptoms are headache loss of appetite drowsiness insomnia fatigue and dizziness caused by the absorption of toxic material from bacterial decomposition Treatment will depend upon the cause X ray examination may aid greatly in the diagnosis Various other studies including stomach contents stool blood and urine will also serve a useful purpose Proper diet medication intestinal irrigations are prescribed as indicated

The small intestine is also prone to benign and malignant growths and bleeding from these growths or Meckel s diverticulum may occur and cause confusion

XVIII THE ACUTE LARGE INTESTINAL SYNDROMES

The normal function of the colon is chiefly concerned with the absorption of water chemical salts and certain enzymatic complexes most of the water is removed from the intestinal contents in the ascending colon together with a number of products of bacterial action Both organic and inorganic salts as well as some nutrient residues and drugs are taken up in the lower portions of the large intestine It is doubtful that much absorption occurs in the sigmoid and below rectal injections are carried by reverse peristalsis upwards into the absorption area of the descending colon

When there is a partial obstruction within the colon as for example from hard feces some of the gas may be forced past the side of the obstruction and there is a tendency for it to accumulate in the region of the cecum the left colic flexure and rectum These accumulations by interfering with neighboring organs may give rise to puzzling symptoms Sometimes remote effects on organs like the heart or gall bladder may lead to erroneous diagnosis It is also possible that blockage in the colon may produce secondary stasis in the small intestine Toxemic symptoms may be relieved quickly when the emptying of the colon removes intestinal stasis Other causes of distention are pneumonia following

abdominal operations peritonitis, or infectious diseases At times distention may be so great that breathing is interfered with by pressure of the distended colon on the diaphragm

Spasm of the colon (irritable unstable or spastic colon) is a common complaint, it is most prevalent in nervous and high strung individuals Anxiety fear, resentment, are often the provocative factors in initiating these spasms with diarrhea or constipation and belching and retained gas may be the result The best treatment in these individuals is to avoid all tension if this is impossible a mild antispasmotic combined with a sedative may be useful

Treatment The application to the abdomen of hot compresses hot water bags or electric pads will relieve the milder cases limitation of fluids the insertion of a rectal tube and a mild antispasmodic may also be tried However in the more severe cases where vomiting collapse and shock are evident nothing should be given by mouth If the diagnosis does not warrant immediate surgical interference a duodenal tube or a Wangensteen or Miller Abbot tube may be inserted through the nose or mouth into the stomach and small intestine in order to decompress the distended gut

HIRSCHSPRUNG S DISEASE

Hirschsprung s disease (idiopathic dilatation of the colon) is believed to be congenital in origin and the usual area of the colon affected is the sigmoid flexure or the entire descending colon occasionally the entire colon is involved It is more common in children, rare in adults In acute episodes the abdomen is markedly enlarged and distended constipation and gaseous accumulations become progressively worse

because of the inability to force out the contents Stool may accumulate in the dilated part of the bowel for days or weeks and give rise to autointoxication or the stool becomes hard through dehydration and may cause obstruction in various degrees Severe colic and discomfort is frequent when the bowel has not been emptied for a long time The stool when passed is extremely offensive in odor because of putrefaction and upon examination may reveal blood and pus probably the result of intestinal wall irritation X ray studies with barium orally or by enema after a partial removal of the impacted stool reveals the pathology

Treatment is directed toward emptying the bowel and attempting to activate the dilated and diseased segment In failure to remedy the condition surgery may be necessary

Acute Constipation Due to Other Causes In congestive heart failure and in some hypertensive patients constipation may be obstinate in addition there are other factors involved and some of these may be due to anoxia causing lowered oxygen pressure which depresses the activity of the muscles of the colon Inflammation of the pelvic and abdominal viscera appendicitis cholecystitis peritonitis may also reflexly cause constipation

Pressure exerted by fibroids of the uterus ovarian cysts hypernephroma and pregnancy may produce constipation through the mechanical effects of narrowing the lumen of the colon by compression or through viscerovisceral reflexes Glomerulonephritis and hydronephrosis may cause reflex ileus without direct pressure effects through stimulation of the nerve supply in the distended or inflamed kidney capsule

TUMORS OF THE COLON

Benign and malignant growths of the small and large intestine and rectum are often overlooked in the early stages Patients who are constipated or alternate with diarrhea with or without blood require careful physical and x ray studies of the gastrointestinal tract *Physical examination of the abdomen* includes digital examination of the rectum and proctosigmoidoscopy and biopsy if indicated More than 75 per cent of all rectal cancers are within reach of the index finger The early symptoms of cancer of the colon and rectum are not distinctive In general there is an alteration of bowel habits unexplained weakness, pain nausea and bleeding from the rectum

Polyp in the lower bowel and the colon may also cause bleeding They may be benign or malignant When seen through the sigmoidoscope may be removed by cautery In all events a biopsy should be done Cancer of the cecum and ascending colon show early signs of anemia when compared to the descending colon sigmoid and rectum Pain while present is usually not severe A careful roentgen study under the *fluoroscope while the barium enema is allowed to flow in slowly will often yield* valuable information regarding the state of the colon Where the diagnosis is uncertain air should be insufflated into the colon with the flow of barium or after the patient is permitted to evacuate

ENDOMETRIOSIS

An often unrecognized acute lesion of the intestine *which* is overlooked because of ill defined symptoms and inaccessible

location is endometriosis of the intestine It usually occurs in women between thirty and fifty most of whom have either not been pregnant or have not carried a child through a full term The symptoms vary with the location of the involved area Acute abdominal pain constipation or diarrhea bloody discharge from the rectum and spasm are the usual complaints

Another unrecognized colopathic syndrome is plumbism When undiagnosed colon disturbance is present one must think of possible lead as a factor in producing colic and recurring symptoms of irritable colon Roentgenoscopy may show acceleration in the passage of the motor test meal through the gastrointestinal tract Signs of spastic or hypertonic colitis and increased porphyrinuria may be present The blood smear shows hypochromia and the number of red blood cells with granular basophilic degeneration is high The gums may show the typical blue line characteristic of lead intoxication

The treatment is that of plumbism plus the anemia

XIX THE ACUTE APPENDICEAL SYNDROME

Acute lower abdominal conditions which often are mistaken for acute appendicitis may be cecal involvement and terminal ileitis mesenteric glands genito urinary tract infections especially in children (pyelitis) in male adults right seminal vesiculitis in females ovarian and tubal involvement ectopic pregnancy torsion of the cecum and mesentery thrombosis of the abdominal vessels intra abdominal bleeding abdominal pregnancy and rupture of the uterus

Other causes for right lower quadrant pain may be in the cecum itself (typhlitis perityphlitis) There is no question that pain in the right iliac fossa can arise in the cecum This pain may *be due to gaseous distention* which is easily *recognized* by the presence of tympany on percussion and gurgling on palpation on auscultation of the abdomen excessive borborygmi are heard

In elderly patients cecal distention may signify chronic intestinal obstruction due to carcinoma of the pelvic colon Soft feces in the cecum associated with constipation may also cause right iliac fossa pain On palpation a characteristic doughy feeling is detected Cecal spasm secondary to colospasm will present marked tenderness over the cecum and often over a rigid pelvic colon in the left iliac fossa Malig

nant disease of the cecum and/or of the ascending colon seldom manifests pain in the early stages

Recurrent or chronic pain in the right lower quadrant of the abdomen may also be due to involvement of the terminal ileum (regional ileitis) and the attacks may closely resemble appendicitis but in the chronic form it produces symptoms of chronic small intestinal obstruction Young adults are usually affected and loss of weight due to diarrhea and temperature is of frequent occurrence On palpation the examining fingers will encounter a thickened terminal ileum resembling a tumor In tuberculous enteritis similar episodes occur but with absence of the tumor mass

Enlargement of the ileo cecal glands following throat infections in children may simulate inflammation of the appendix Tuberculous glands involving the right lower abdomen may be difficult to differentiate from non specific mesenteric adenitis In both conditions pain may occur in the iliac fossa or around the umbilicus No shifting pain although tenderness and muscular guarding without true rigidity as in appendicitis may be present but vomiting is rare Differentiating between true tuberculous and non specific adenitis in children may be made by a positive Mantoux reaction and a pale fatty stool

Involvement of the urinary tract in adults and children may cause pain in the right iliac fossa Chronic pyelitis stone in the lower ureter hydronephrosis inflammation of the prostate and/or the right vesicle may cause frequency of voiding pus cells crystals red blood cells and bacilli are found in

the urine A stone in the ureter may cause pain to radiate from the loin to the groin, impaction of a stone in the lower third of the ureter or kinking of the ureter localizes the pain in the right lower quadrant Chronic or intermittent hydronephrosis may obscure the symptoms and add to the confusion in differential diagnosis The examiner must remember that in appendicular involvement the pain begins in the upper abdomen before localizing in the right iliac fossa also that nausea precedes the vomiting Although in children the initial manifestation when acute pyelitis is causing the trouble is vomiting with abdominal pain and rise in temperature 102 104° F Obtaining a catheterized specimen of urine will help to obviate the error

Careful bimanual palpation of the loin and complete investigation of the urinary tract should aid in the differential diagnosis

GYNECOLOGIC CONDITIONS

In adolescent girls and child bearing women gynecologic conditions are fairly common causes of pain in the right iliac fossa Acute appendicitis may be confused by bleeding into the peritoneal cavity from a ruptured Graafian follicle or corpus luteum It may also mimic pregnancy or abdominal conditions requiring surgery These ruptures of the ovarian follicle occur most frequently in the late teens or early twenties and athough no specific relation to occupation can be determined the frequency is almost twice as great for unmarried as for married women Trauma or exertion does not seem to be a causative factor although the pain occasionally starts during sexual intercourse

The chief symptoms caused by a bleeding ovary is pain

which ordinarily begins and persists in the lower abdomen most frequently on the right side and is usually more severe than in appendicitis A great many patients recall that in appendicitis the pain began in the epigastrium later localizing in the right lower quadrant Shoulder pain occurs rarely even when fluid extends upward and produces diaphragmatic irritation Nausea present in most cases is accompanied by vomiting in about twenty five per cent of the patients

Although right lower quadrant tenderness is found in almost nine tenths of the patients only about one fourth have definite associated muscle spasm Adnexal tenderness is frequently found on pelvic examination but a mass is seldom palpable Leukocytosis usually occurs though seldom severe and the temperature is rarely elevated above 101° F with only slight increase in pulse rate

Preoperative diagnosis is difficult, although the relation of pain in the abdomen and the rupture of a Graafian follicle midway in the menstrual cycle is an important clue in differentiation Rupture of a follicle for ovulation occurs in most women between the ninth and twentieth days of the cycle in some patients between the eleventh and fourteenth day Knowledge of the habitual length of the cycle is of some value since coincidence of symptoms and established time of ovulation may aid in the diagnosis and obviate surgery

Bleeding from the ovary not associated with ovulation may be caused by rupture of a corpus luteum or a corpus hemorrhagicum or a retention cyst of the ovary with bleeding from the ovarian surface at the time of a menstrual period At operation the surgeon may find an accompanying acute inflammation of the appendix or a Meckel s diverticulum

INTRAPERITONEAL CAUSES OF PAIN

PAIN as a result of—	COLIC	HEMORRHAGE	OBSTRUCTION & TORSION	PERFORATION	INFLAMMATION
a Onset	abrupt	abrupt	acute	dramatic	varies
b Character	griping crampy rhythmic	steady dull severe	severe griping colicky crampy	agonizing severe and steady	varies with organs involved
c Location	over anatomical unit	over area	about or below umbilicus	over involved area	over involved organ
d Duration	intermittent rhythmic	steady continuous	rhythmic	continuous steady and rhythmic	continuous
e Radiation	typical	none	none	none	none
f Associated Symptoms	repeated vomiting repeated retching restlessness no fever	patient blanched anxious sweating rapid pulse no fever	restless vomiting abdominal distention no fever	won't move lies flat shock	rise in temperature nausea vomiting
g Objective Findings	little or no tenderness no muscular rigidity	peritonism tenderness muscular resistance low r b c low hemoglobin low hematocrit no leucocytosis	no tenderness no rigidity hyperperistalsis visible audible palpable mass tumor *hernia* operation scar distended loop	tenderness rigidity x ray gas bubble	local tenderness muscular resistance leucocytosis rectal examination

Appendicitis during menstruation is a rarity and is often confused with a ruptured enlarged cyst of the ovary torsion of the ovary or a ruptured follicle It is especially significant when lower abdominal and pelvic pain occurs midway during menstrual period the patient may safely be subjected to expectant or conservative treatment and thus avoid needless operation

Ectopic pregnancy is usually preceded by some menstrual irregularity both as to time advancement or retardation spotting or even a profuse metrorrhagia The patient also gives a history of morning sickness nausea vomiting tingling of the breasts salivation may or may not be present There is nearly always complaint of pain or tenderness low in the pelvis which is more marked on one side The patient may also complain of rectal tenesmus pain on bowel movement and bladder irritation

In summary the causes of right sided abdominal pain may be listed in the following order of priority

1 Appendicitis
2 Nephrolithiasis
3 Biliary diseases
4 Peptic ulcer
5 Intestinal disorders
6 Ovarian conditions

XX THE ACUTE GALLBLADDER SYNDROME

Acute cholecystitis usually presents itself as an acute abdominal emergency with right upper quadrant pain that is often referred to the right shoulder when the inflammatory exudate reaches the diaphragm and causes irritation of the phrenic nerve Fever nausea vomiting leukocytosis, rigidity of the abdominal wall especially in the right upper quadrant and palpable gallbladder are the prominent symptoms Jaundice may appear up to 25 per cent of the cases but is usually not severe

The ratio of women to men who develop cholecystitis is *approximately 2 1 and the axiom that a female who is fat fair forty and fecund is usually the victim of gallbladder disease has been changed The management of these acute* cases is a controversial problem whether to operate during *the height of the attack or to wait for the acute stage to sub*side spontaneously or whether the increasing exudation of the gallbladder wall will lead to perforation

Trauma to the abdomen may cause cholecystitis and may become a legal question especially in compensation cases Some patients who sustained accidental injuries to the abdomen without visible external signs developed hemorrhage

from the stomach or the duodenum while others had cholecystic manifestations Occurrence of cholecystitis postpartum has been noted and may be explained on the ground that a severe strain is placed on the liver and gallbladder during pregnancy The incidence of cholecystitis after typhoid is well established there is no question that an infection by the typhoid baccillus can occur when the serous membrane of the gallbladder becomes damaged by biliary stasis

Other types of cholecystic pathology which may occur are suppurative membranous phlegmonous gangrenous fibrous villous or papillomatous glandularis proliferans filtrating masked recurrent gonococcic allergic and non lithic cholecystitis The bacteria most frequently found is the low virulent streptococcus viridans Although spontaneous resolution occurs at times in mild acute infections of the gallbladder it can hardly be anticipated in advanced acute processes with intense and deep seated inflammation of the gallbladder wall abscess formation and necrosis

Bile peritonitis may be due to leakage of bile from the gallbladder The clinical picture is obscure and is not diagnosed before laparotomy At operation the surgeon usually finds an effusion of bile in the lower abdomen but no visible ulcer or appendix perforation however careful examination of the gallbladder will reveal a small insignificant tear

Bleeding from the gallbladder may often be confused wi[illegible] a bleeding ulcer especially if the history is doubtful [illegible] adhesions cause a deformity of the duodenum or pylorus [illegible] simultaneous occurrence of gallstones and ulcer is [illegible] the ulcer may heal after the hemorrhage and onl[illegible]

are found at operation In the presence of a bleeding gall bladder diagnosis should not be difficult if one bears in mind that an anemic appearance demonstrable secondary anemia and a positive guaiac test of the feces of elderly patients do not always indicate cancer because chronic cholelithiasis may also present these findings

The acute attack may subside and chronic cholecystitis supervene When this condition develops the patient complains of vague insidious disorders such as intolerance of fatty foods belching postprandial epigastric distress nausea vomiting pseudo biliary colic or colic due to gallstones which frequently accompany the inflammatory state The differential diagnosis must exclude extra and intra abdominal conditions and cholecystography is advised In a small number of cases of chronic cholecystitis and cholelithiasis carcinoma may develop or the passage of a gallstone into the common duct results in biliary tract obstruction with jaundice and at times acute pancreatitis

Nonsubsidence of the acute attack may lead to pericholecystic abscesses due to permeation of the infection or perforation of the gallbladder peritonitis ascending cholangitis liver abscesses subdiaphragmatic abscess and septicemia When confronted with these complications a careful analysis of the history may aid in establishing the diagnosis Many of these patients whose gallbladders go on to suppuration and perforation present a rigid or fixed right diaphragm and a board like rigidity of the abdomen high temperature increased leukocytosis impairment of resonance and coarse rales in the right lower lobe of the lung These findings are not due to pleurisy and/or pneumonia but are caused by

congestion and upward pressure from pathology below the diaphragm

Adhesions to adjacent organs are of frequent occurrence as a result of repeated inflammation of the gallbladder Roentgenologically and clinically it may be difficult to differentiate between a duodenal ulcer and adhesions however questioning the patient as to pain and hunger rhythm may be helpful

Occasionally inflammation of the gallbladder with or without stones may have referred pain to the left abdomen thus masking the true cause and delay treatment This referred pain may be due to nerve aberration One must also be on the alert for cardiac conditions such as angina pectoris coronary thrombosis and infarction which may give rise to symptoms similar to those of cholecystopathic manifestation Tenderness and rigidity of the liver area may be found because of sudden blood engorgement of the liver Cholecystitis with or without gallstones and acute cardiac conditions may coexist The examination of the blood for transaminase unquestionably will be helpful A high transaminase favors coronary disease

Rare but often missed is transposition of the viscera (partial or entire) with the gallbladder under the left costal area, attacks may simulate angina or coronary disease The true condition is diagnosed roentgenologically

A presumptive diagnosis of neoplasm can be made when with a history of previous cholecystic disease a hard nodular painful and irregularly rounded tumor can be palpated in the right upper quadrant of the abdomen A smooth globu

lar distended gallbladder is not to be confused with the hard irregular mass noted in primary carcinoma

Treatment The patient with acute cholecystitis is to remain in bed he is to apply heat or cold to the affected area he is to get antibiotics, and a liquid diet unless nausea and vomiting is present small sips of water and weak tea later well cooked Farina cream of wheat and fruit juices may be added no attempt should be made to move the bowels For the pain Demerol with or without atropine by hypodermic injection is given the latter is contraindicated when glaucoma is suspected *Morphine should be avoided because it increases nausea vomiting and intraductal pressure,* nitroglycerine grain 1/150 or grain 1/100 sublingually or by injection may relieve ductal spasm A suppository containing 10 grains of aspirin with one grain of codeine may *also be tried for relief* of pain When narcotics are contraindicated or in elderly individuals intravenous injection of 10 cc of a 10 or 20 per cent solution of neo calglucon may relieve the pain It must be injected slowly otherwise a burning sensation over the entire body may be felt This medication may also be used in gall or kidney stone colic

Operation should be avoided in the acute stage it is advisable to wait until subsidence of the temperature unless perforation or empyema of the gallbladder is suspected

XXI THE ACUTE PANCREATIC SYNDROMES

Intestinal disorders in general and intestinal sluggishness in particular play an important role in the etiology of pancreatic disease Intestinal loss of motility involving the duodenum may cause stagnation of pancreatic secretion Extension of inflammation of the duodenum into the pancreatic ducts or infection of the latter by invasion of intestinal bacteria along the column of stagnant pancreatic juice may also be factors Disturbances of the hormone mechanism through pathological changes in the duodenal mucosa and circulatory disturbances caused indirectly by enlargement of the liver with duodenal atony are not infrequent findings Spread of infection via the lymphatics from a cholecystitis or a choledochitis into the pancreas and organic involvement of the pancreas through the liver is less common

Pancreatitis may occur at any age but is most frequent during middle life It is uncommon in the first two decades except as a complication of mumps

Classification Considerable difficulty is presented by the problem of classifying the pancreatic diseases into clinical entities They may be divided into acute and chronic affections into medical and surgical diseases or they may be con

sidered from the inflammatory and neoplastic point of view or as acute pancreatitis of the hemorrhagic gangrenous suppurative types

In chronic pancreatitis all forms of sclerotic changes of *the gland* such as those associated with gallstones jaundice or cirrhosis occur Tuberculous infection may produce sclerosis and rarely caseation Syphilitic infection may result in sclerosis or gumma Cysts and pseudocysts are common at times cysts may form after trauma Foreign bodies in the duct or ampulla (may be calculi) are seen New growths like adenoma fibroma and sarcoma (all rare) carcinoma (primary or secondary) of head body tail or ampulla of Vater are found

ACUTE HEMORRHAGIC PANCREATITIS

The most important and urgent disturbance of the pancreas is acute hemorrhagic pancreatitis a syndrome which has always carried a serious prognosis The most frequent cause of acute hemorrhagic pancreatitis is infection of enterogenous origin with Bacillus Coli but bacterial invasion by other *pyogenic organisms may take place through the blood* stream The entrance of bile into the pancreatic duct due to gallstones near the ampulla of Vater is at times a precipitating cause

Symptoms Abdominal pain is the most constant complaint occurring in about 90 per cent of patients with acute pancreatitis The attack which is sudden and violent may occur in individuals who have previously enjoyed apparently good health The severe vomiting and excruciating pain in the epigastric region resembles peritonitis Collapse and death

appear imminent or there may be transient improvement followed by a new attack

Physical examination is difficult but when palpation is possible hardness and a tumor mass may be felt in the upper abdomen this may be the enlarged pancreas

The syndrome may simulate a perforating duodenal or gastric ulcer gangrenous perforation of the gallbladder or retrocolic appendix intestinal obstruction in the upper abdomen pyelitis renal or adrenal lesions ectopic rupture mesenteric or splenic thrombosis aortic aneurysm and rupture mesenteric arteriospasm acute cardiac dilatation and collapse in angina pectoris and violent poisoning by food or chemicals A very important sign if present is a bluish discoloration of the umbilicus (Cullen s sign) when ectopic rupture occurs Necrosis intensifies the pain nausea and vomiting tympanites and shock are characteristic in acute pancreatitis Vascular destruction results in extensive bleeding and infiltration the escaping pancreatic juice produces autodigestion Individuals who are addicted to alcohol have a tendency to pancreatic necrosis with fatal results

PURULENT PANCREATITIS

Acute and subacute purulent pancreatitis may result either from an extension of the suppuration of adjacent organs or through invasion of pus producers through the blood or lymph The infection may be an ascending one from the intestines especially in cholelithiasis

Symptoms The symptoms resemble those of hemorrhagic pancreatitis but develop much more slowly are milder and not followed by collapse The onset is characterized by alter

nating diarrhea and constipation Functional tests of the pancreas may indicate mechanical disturbance There may be resistance on palpation corresponding to the diagonal position of the enlarged pancreas

SUBACUTE EDEMATOUS PANCREATITIS

Subacute pancreatitis or so called acute edema of the pancreas may be secondary to an acutely inflamed gallbladder The symptomatology is important sometimes without warning the patient is stricken by an intensely violent acute abdominal pain and is often moribund before the cause of the disturbance is even suspected and before there is opportunity for operative interference

Symptoms Unless there is predominating or complicating active disturbance in the gallbladder or bile ducts the picture is rather characteristic One may trace a definite history of recurrent attacks of pain extending over a period of months or years This however may vary as in some cases there are no previous attacks The severity and duration of these pains subside spontaneously and are often thought to be due to an attack of biliary colic or gallbladder inflammation This is probably true in some cases until the serious acute involvement occurs or until chronic pancreatitis is established

These attacks are rather of a specific character differing decidedly from the usual gallbladder pain and at operation the gallbladder is found relatively uninvolved The patient complains of excruciating deep boring pain in the mide pigastrium, with radiation to the left In addition there is tenderness on deep pressure in the epigastrium with very

slight if any rigidity Often there is a definite area of hyperes thesia (Head zone) in the left flank at the level of the eighth to the tenth dorsal vertebrae Jaundice is usually absent unless there is a calculous or inflammatory common bile duct obstruction the icterus index is not increased A moderate elevation of temperature and pulse rate may occur a mod erate leukocytosis a trace of sugar and a decreased sugar tolerance at times are also noted Examination of blood amylase during the acute stage will show marked elevation which tends to return to normal after twenty four or forty eight hours

The determination of the antithrombin level either as a supplement to amylase determination or in borderline titers is considered positive during the first twenty four hours after the acute onset however the antithrombin level is nonspecific in known or suspected pancreatic carcinoma

Opiates do not alter the antithrombin level while neostig min is antagonistic and invalidates the test

Pregnancy may be a forerunner or complicating factor of acute pancreatitis When confronted with a pregnant patient with severe upper abdominal pain increased nausea vomit ing collapse and rise of pulse and temperature acute pan creatitis should not be omitted from a possible etiology During pregnancy there is an increase in pancreatic function with elevation in lipase and trypsin concentrations and rise of serum amylase and urinary diastase When pancreatic in volvement is suspected the above tests may be of material help in differential diagnosis

The use of morphine Demerol or other narcotics may mask the symptoms and lead to erroneous diagnosis

ACUTE PANCREATIC DUCT LITHIASIS

Stone formation is possible in both the main and smaller pancreatic ducts calcium deposits within the gland substance must be differentiated from freely moving stones Changes in the chemical and enzymatic composition of the pancreatic juice may result in the precipitation of calcareous phosphate salts when these discrete precipitates are less than 1 mm in the largest diameter they may pass freely into the intestinal cavity but larger stones may cause partial or complete obstruction The Wirsung s duct joins the common duct at the papilla of Vater where both enter the duodenum In a small percentage of cases Santorini s duct draining a portion of the head of the pancreas enters the duodenum proximal to the papilla of Vater

Chronic Pancreatic Lithiasis This is a relatively rare clinical condition and the symptoms are not characteristic Stones in the pancreas are often associated with stones in the gall tract they are easily overlooked at operation for gallstones A few stones may be visualized by x ray when special care is used in film interpretation A stone within the pancreatic duct is more common in older individuals and is believed to be due to a previous infection of the duct of the pancreas with some obstruction in the outflow of pancreatic juice into the intestine The stones are white and occur in groups rarely single When the pancreatic duct or its branches are obstructed by stones the channel behind the obstruction becomes dilated and in some cases this results in cyst formation Inflammation of the pancreatic substance occurs when backward pressure of the fluid increases

Symptoms The symptoms center around manifestation of

pain in the epigastrium it may be spasmodic or continuous and varies in severity At times it is intense and can hardly be controlled even by hypodermic injection with opiates The pain often radiates to the left abdomen and left shoulder blade and is frequently mistaken for gallstone colic perforated gastric or duodenal ulcer The shock from the pain occasionally results in general collapse In some cases when the bile flow is interfered with jaundice may occur On examination the abdomen is quite tender to pressure and the pancreas may sometimes be felt when it is swollen The stool often reveals a large quantity of undigested fat and meat fibers and the urine may show the presence of sugar The acute symptoms may subside and the stones are often passed and may be found in stool examination

TREATMENT

The treatment is symptomatic Medical measures including bile drainage have been tried from time to time with variable degrees of success A limited diet with a low calcium content and pancreatic preparations to aid digestion are indicated When medical measures fail surgery shoud be considered for removal of the pancreatic block

In some instances because of a damming up of the pancreatic secretion part of the gland substance may undergo fatty necrosis due to the digestive action of the fat ferment lipase It is believed that the fat ferment of the pancreatic secretion is activated by the presence of bile in the pancreatic duct At times because of backward pressure of the pancreatic juice an abscess of the pancreas may develop but this may also follow in the course of any inflammation of the pancreas

Symptoms will depend upon the primary condition which usually precedes the involvement of the pancreas As indicated previously such ailments are characterized by various digestive symptoms Pancreatic involvement however focuses attention on disturbances of intestinal digestion for it is the pancreatic juice which predominantly acts upon the foods in the small intestine during the process of digestion evidence of disturbed pancreatic function is found on fecal examination Withdrawal of the duodenal contents will also most likely show a diminution in the various ferments comprising the pancreatic juice In the simple cases the symptoms will be chiefly those of a mild indigestion but in the severe forms with complications symptoms become aggravated and in addition pain vomiting, diarrhea or jaundice may appear When fatty necrosis occurs the symptoms are increased and simulate those of acute hemorrhagic pancreatitis In abscess formation the symptoms and the course of the ailment will vary depending upon whether the abscess is acute or chronic When the abscess is large it is easily felt during the abdominal examination

The treatment is directed mainly toward improving the outflow of pancreatic juice thus lessening stasis within the pancreatic ducts reducing the quantity of secretion elaborated by the pancreas may also be important Atropine belladonna and anticholenergic drugs may be given to retard pancreatic and biliary secretion and reduce spastic manifestations Epsom salts or cathartic waters may also reduce the degree of pancreatic secretion Bile drainage simple bland liquid or semisolid foods containing virtually no fats are also indicated Failure to relieve the patients by medical means or when complications prevent carrying out a medical regimen are indications for surgery

Part III

THE PULMONARY EMERGENCIES

George Guttman Ornstein M D F A C P F A C C P D I M

XXII SPONTANEOUS PNEUMOTHORAX AND LUNG COLLAPSE

Pneumothorax may occur spontaneously It usually occurs idiopathically and spontaneously in apparently healthy young males and at an age most often between twenty and forty though it may occur in any age It also may occur occasionally in females and more frequently than is suspected but the occurrence in males still outnumbers the occurrence in females

The diagnosis is chiefly based on the symptoms physical findings and roentgenograms of the chest The findings are the same in both the male and female "Idiopathic pneumothorax is frequently found in healthy individuals At one time spontaneous pneumothorax was only associated with the incidence of pulmonary tuberculosis because the latter disease left the lung weak near the apical pleural areas Pneumothorax occasionally occurs in pulmonary tuberculosis because these weak areas especially in healed apical pul

monary tuberculosis and an associated emphysema of the lung close to the pleura may cause an attack of pneumo thorax Again we say it may be a cause but it is very in frequent

Causes of Pneumothorax When idiopathic pneumothorax occurs in young adults the chief cause is due to a ruptured pleural bleb These blebs are not congenital as formerly be lieved but develop when the diaphragms are fixed with the glottis closed and when considerable pressure is developed air is forced into the upper lobes The upper lobes are dis tended and this forceful entrance in the upper lobes over distends pre existing blebs and ruptures the elastic fibers *in the sub pleural alveoli The blebs are usually the result* of over distention of the lobes and frequently can be visual ized in the apices of both lungs when these blebs become greatly over distended they may rupture into the pleura and cause a pneumothorax

There is a phase of life of young adults when they pass through a growing period and during these periods the upper lobes are much distended Production of these em physematous blebs which may occur bilaterally and chiefly in the apices of the upper lobe may be the cause of the oc currence of spontaneous pneumothorax They may occur on one side and subside but it is known that it is common for blebs to be in both lungs and therefore, we hear of the occurrence of a spontaneous pneumothorax in one lung *which may subside and then a reoccurrence* of the same dis ease over the opposite lung When a bleb is formed a check valve is formed over the bleb and air can enter and distend it when it becomes over distended it bursts It frequently

happens that a bleb may rupture while the patient is in bed or asleep the walls of the bleb may be so thinned out that it easily ruptures Therefore we must say that an idiopathic spontaneous pneumothorax in patients may occur because of the continual thinning out of the wall in obstructive em physematous blebs and this sudden rupture may occur at any time of the day or night without history of exertion It is the check valve obstruction mechanism associated with emphysematous blebs that plays a most important role in the pathogenesis of the disease

Pathogenesis Inasmuch as most cases of spontaneous pneu mothorax occur in healthy individuals there is scant autopsy material The pathogenesis of spontaneous pneumothorax previously has been based on very few autopsy findings and in these cases emphysematous blebs were not demonstrated Usually the blebs found in these patients were produced by scarred lungs where there was a weakness in the areas de veloped by the pathology in these areas an associated dis ease of the pleura was found Since tuberculosis is a common disease spontaneous pneumothorax is frequently found where healed tuberculosis is noted with its close association with the adjacent weakened pleura From the early literature a distinct impression was given that most cases of idiopathic spontaneous pneumothorax were due to some form of healed tuberculous disease The extent of the pneumothorax depends on the size of the emphysematous blebs and the elasticity of the adjacent lung tissue When most blebs rupture the pneu mothorax may be very extensive The signs of a pneumothorax vary usually there is a sudden pain in the chest dif ficulty in breathing and often a dry hacking cough The pain

varies considerably and may often resemble an acute coronary occlusion Dyspnea may be present and chiefly increases as the pneumothorax becomes larger When the patient begins to walk dyspnea frequently increases The pain may often be referred to the shoulder or the diaphragm There may be signs of shock but pain and dyspnea are chiefly present and both are increased with any motion Some times spontaneous pneumothorax may occur with very little acute pain The pain may be limited and may only radiate to the shoulder

Pneumothorax is frequently overlooked However the physical findings are always specific On physical examination there may be hydro resonant signs over the involved area and because of the pneumothorax the breath sounds may be absent or markedly diminished On the other hand over the contralateral lung breath sounds are exaggerated

X ray Examination The most important procedure in diagnosis is fluoroscopy and x ray Usually the fluoroscope examination reveals the pneumothorax if it is of any size It can easily be observed in the roentgenograms of the lungs During inspiration the lung may have partially re expanded and the presence of the pneumothorax been overlooked it is necessary to take a roentgenogram of the lung during expiration In expiration a small pneumothorax may be discovered because in expiration a space may be seen between the lung and the pleura on the involved side

A history of sudden pain in the thorax accompanied by dyspnea is very suggestive of pneumothorax

Prognosis In patients with idiopathic pneumothorax the prognosis is excellent In our experience when it occurs in

apparently healthy young adults there has been no development of pulmonary tuberculosis Patients frequently have recurrences because blebs may occur near the pleura usually bilaterally and any bleb may develop a check valve mechanism with a retention of air and distention Because of the check valve mechanism the bleb may distend and ultimately rupture through to the parietal pleura and produce a pneumothorax Patients with many recurrences have a favorable prognosis

TREATMENT

After recognition of the pneumothorax treatment is very simple The patient is put to bed and kept there until the lung re expands Small doses of codeine may be given for control of the cough if present Constipation must be avoided straining at stool may cause a relapse Patients should even be cautioned against laughing since intrapulmonary pressure may be increased The lung usually re expands in four to six weeks

When a tension pneumothorax occurs pressure may be relieved by inserting a catheter into the pleural space The free end of the catheter is placed into an open bottle containing a few inches of water air bubbles should pass through the water

A recent surgical method especially in recurrences of spontaneous pneumothorax is the insertion of substances into the pleural cavity which produce adhesions of the visceral and parietal pleura However the whole pleural space may be eliminated and bleb formation which previously could rupture into the pleural space may go across the lung to the mediastinum accompanied with much greater danger

than if it had entered the pleural cavity alone Hence this may not be a safe procedure

TRAUMATIC PNEUMOTHORAX

Traumatic pneumothorax may develop in several conditions it may occur as a result of gun shot or stabbing accidents falls automobile injuries crushing injuries of the chest or any condition where the pleural cavity is involved Suspect such a condition when a rib is broken for it may puncture through the visceral and parietal pleura In bronchoscopy and esophagoscopy and in renal operations or sympathectomies a pneumothorax may be produced In pneumothorax where there is increased pressure, the heart and mediastinum may be misplaced and treatment is chiefly the removal of *the air causing the tension pneumothorax*

ATELECTASIS OR COLLAPSE OF THE ALVEOLI OF THE LUNG

Atelectasis is anatomically a collapse of the alveoli of the lung and it occurs more commonly than is generally believed It develops frequently because branches of the bronchial tree to the lung tissue are blocked, the cause of this is manifold For example bronchial secretions or tumors or even mechanical obstructions are common causes While the etiology of atelectasis varies the specific cause of such blocking must be determined when the cause is removed the lung usually re expands

The cause of the obstruction should be investigated and *if an etiological* factor such as tuberculosis causing an atelectasis is not found the bronchial tree can be investigated by means of bronchoscopy the actual cause of the blocking can usually be quickly determined For example a solid ob-

ject like a peanut could cause a blocking obstruction and produce atelectasis of the lung If food is aspirated it could easily cause a block of the bronchus peculiar phenomena such as obstruction emphysema may be the end result of food aspiration

If fragments of food should cause obstruction it may be accompanied by inflammation and infection which in them selves produce an inflammatory condition of the mucous membrane of the bronchus which may block the bronchus leading to a portion of the lung

A six year old child who had previously been treated for allergy had a quarrel with his sister from whom he snatched a bag of peanuts she pursued him and before he was caught he threw a handful of peanuts into his mouth After a small choking episode he had a return of his asthmatic attacks The left lung did not expand as fully as the right lung During expiration air escaped from the normal right lung but could not escape from the left lung because of the block in the main bronchus due to the aspiration of the peanut The left lung appeared to be as fully extended as the right lung this was noted during fluoroscopy The left lung was dull over the upper lobe and the breath sounds were diminished in in tensity Over this same site wheezing rales were heard The mediastinum had moved toward the left and because of the history of the sudden swallowing of peanuts aspiration of a peanut was suspected The patient was immediately bron choscoped and a peanut recovered followed by disappear ance of all symptoms

Hemorrhage This may be a cause of atelectasis but when the blood is absorbed the atelectasis disappears This may

occur in a great many conditions when we think of hemor rhage the most frequent cause is pulmonary tuberculosis Here the sputum examination may be positive for tubercle bacilli and the explanation for the cause of the hemorrhage and possibly the cause of the atelectasis may be suspected

Symptoms One of the frequent complications of pulmonary infarction is atelectasis the extent of the pathology will de pend upon the bronchi involved In such cases the atelecta sis is very apt to be demonstrated by physical signs Breath sounds in the lung involved are absent the mediastinum and heart are pulled toward the side of the thorax involved and the diaphragm on that side is usually high When atelectasis occurs with pulmonary emboli there are many symptoms especially where dyspnea tachycardia and cyanosis occur and where respiration is increased also where prostration is great

Respiratory Movements The chest on the affected side looks flat and narrowed There may be depressed intercostal spaces and usually the respiratory movements are diminished or absent Sometimes this condition is associated with pleural fluid Frequently the tubular breathing which may be heard over the flat note is caused by sound transmission over the pleural effusion of an underlying atelectasis

Pulmonary Carcinoma This should always be considered as a possible diagnosis Even when tubercle bacilli are found there may be a co existent tuberculosis and carcinoma It should be recalled *that most carcinomas of the lung arise from the mucous membrane of the bronchial tree* In periph

eral carcinoma absence of breath sounds is very common and in the main bronchi it may take a long time before growth of the tumor blocks the airway In early carcinoma of the periphery of the lung diminished breathing is one of the chief signs Wherever diminished breathing occurs as in atelectasis it should be remembered that such a condition may conceal the occurrence of pulmonary infarction It has been our experience that where a diagnosis of atelectasis has been made the pathologist very often finds a pulmonary in farction the latter condition is frequently masked by the diagnosis of atelectasis With the blocking of a bronchus the most important differential diagnosis is pulmonary car cinoma A patient may have been bronchoscoped and the carcinoma missed

Tuberculosis Next in importance is the diagnosis of pulmo nary tuberculosis However it is important to remember tu-berculosis and carcinoma may co exist in the same lung and the presence of tuberculosis does not rule out the possibility of a carcinoma It is wise to remember that in primary tu berculosis a large gland may block the major bronchi and cause atelectasis

In summary it is recognized that there are many factors which cause an atelectasis and which involve part of a seg ment or even the whole lung The etiology of this atelectasis must be studied there are many factors and the exact cause must be determined When atelectasis occurs there may be sufficient ventilation for an individual to carry on if there is no progression of the disease

XXIII PULMONARY HEMORRHAGE

Pulmonary hemorrhage should not be classified by the quantity of blood coughed from the lung it may be a glassful or more or it may be very much less There was a time when pulmonary hemorrhage was closely associated with pulmonary tuberculosis one of the great authorities in tuberculosis stated that hemorrhage should be a tablespoonful or more in order to indicate the possibility of pulmonary tuberculosis This was an unfortunate definition of pulmonary hemorrhage Under this concept physicians would inquire whether a tablespoonful or more was coughed from the lungs before venturing a diagnosis If the blood raised was less than a tablespoonful a diagnosis of pulmonary tuberculosis was not considered likely Later any evidence of blood coming from the lungs was thought to be a tuberculous lesion of the lung Any evidence of blood whether it is seen alone or mixed with sputum is now considered as an *hemoptysis* The important diagnosis is to determine where the blood comes from Pulmonary tuberculosis at one time was the most common cause of hemoptysis or blood tinged sputum Even today with a marked decrease in pulmonary tuberculosis hemoptysis or blood tinged sputum is a common finding in this disease

Next to tuberculosis the most common pulmonary disease is carcinoma of the lungs When hemoptysis or blood tinged sputum is noted in the history a diagnosis of pulmonary tuberculosis is considered first and pulmonary carcinoma next

Coughing of blood may come from many diseases of the lungs and is frequently found in bronchiectasis and infarcts of the lung It may occur in pulmonary abscess but not as commonly The important finding in pulmonary abscess since most of these infections are aerobic is the foul odor associated with the cough and expectoration but hemoptysis may also occur in these conditions One of the causes of hemoptysis frequently overlooked is bloody sputum associated with an acute hypertension where the lung is involved Hemoptysis or any bleeding from the bronchi may be associated with cardiac disease in pulmonary pathology caused by parasitic infections Blood spitting is a frequent discovery in Friedlander s pneumonia with its peculiar cherry red colored sputum In years past when syphilis was common one occasionally saw latent syphilitic conditions where a laceration of the larynx in the third stage of the disease may cause bleeding the blood coughed up may suggest origin in the lungs Ulceration in the larynx trachea bronchi and a ruptured aneurysm may cause hemoptysis In crushing injuries of the chest blood may cause an obstruction of the air ways The presence of pulmonary hemorrhage demands immediate intensive investigation of the lungs

When hemoptysis occurs an x ray of the lungs may disclose a cavity if a single cavity is seen in the lung hemorrhage can be controlled by pneumothorax In view of the recent knowledge concerning anti tuberculous drugs it is

considered wise to provide chemotherapy at the time pneumothorax is instituted With collapse therapy both antituberculosis *chemotherapy and antibiotic therapy in the form* of streptomycin should be given, experience has shown that this combination is more successful than when only antituberculosis *therapy is used When pneumothorax cannot* be done a pneumoperitoneum plus chemotherapeutic drugs should be instituted at the same time

Pulmonary stones (usually called primary complex) are not uncommon in the hilar regions in chest films Our experience *has shown that rotation of the patient during the fluoroscopic* examination may reveal changes in the root areas not seen in the conventional x ray taken in the posterior anterior position *When a stone ruptures into a bronchial artery the* subsequent hemoptysis may be severe Such a diagnosis is more likely when the patient expectorates a pulmonary stone If a diagnosis of pulmonary tuberculosis cannot be made carcinoma of the bronchi must be considered A diagnosis of pulmonary tuberculosis or any other type of pulmonary pathology does not necessarily rule out malignancy Until a diagnosis of pulmonary malignancy can be completely eliminated it must be suspected The discovery of tubercle bacilli in the sputum is naturally very helpful in the diagnosis of pulmonary tuberculosis although a negative sputum does not necessarily rule out pulmonary tuberculosis

Bronchoscopy Inasmuch as carcinoma of the lung usually takes its origin from the mucous membrane of the bronchus the bronchial tree should be carefully investigated The trachea and the bronchi are extensive areas for *investigation*

and it is not uncommon for a number of bronchoscopic examinations to be made with negative findings In actual practice tomograms of the lungs in the anterior posterior views from 4 cm to 14 cm should be made before bronchoscopy If suspicious lesions are noted the bronchoscopist can now investigate these areas and biopsies can be secured

Bronchiectasis Hemoptysis is common in bronchiectasis and has a tendency to recur A diagnosis of bronchiectasis should be confirmed by a bronchogram Dilatation of the bronchi is common after bronchiectasis of long duration Recurrence of hemoptysis is a common occurrence in bronchiectasis It should be noted that pulmonary carcinoma also occurs in patients with bronchiectasis Carcinoma is frequently noted in such instances during the bronchoscopic procedure Narrowing and rigidity of the bronchi is not uncommon in neoplastic disease

TREATMENT

The treatment of pulmonary hemorrhage depends on the etiology of the disease Should the hemorrhage be severe therapy may have to be initiated immediately Otherwise diagnosis may be established and then treatment begun To control bleeding in pulmonary tuberculosis pneumothorax is frequently inducted or pneumoperitoneum if the situation prevents the induction of a pneumothorax If both these methods fail then the modern chemotherapy is given, which consists of streptomycin one gram twice weekly intramuscularly and daily doses orally of isoniazids and PAS Usually isoniazid is given in the dosage of 4 mg per kilo daily in four divided doses and PAS 18 grams in divided doses three

times daily This therapy may be sufficient to stop the hemorrhage and clear up the tuberculous disease

When hemoptysis is small a blood coagulant such as Ceanothyn may control the situation

Hemorrhage may occur in many situations and collapse therapy is used when the bleeding is severe Treatment will depend on the diagnosis of the etiology of the disease

XXIV ACUTE BRONCHIAL ASTHMA AND BRONCHO PULMONARY ALLERGIES

Asthma is a disease characterized by spasmodic attacks of wheezing and coughing caused by irregular periods of con traction of the bronchi With these attacks of wheezing there may be periods of dyspnea and they are most frequently allergic in origin It is not unusual for these attacks of cough wheezing and dyspnea to be diagnosed as allergic asthma Aspiration of a foreign body into the bronchus or a division of the bronchial tree will produce the same symptoms These symptoms may also occur with fibrosis or pressure which diminishes the lumen of the bronchus Therefore we may have symptoms of cough wheezing and dyspnea and not have an allergic background Obstructive emphysema may be accompanied by these symptoms and this condition is usually suspected to be asthma

Fluoroscopy and X rays X rays taken on inspiration and ex piration will reveal a shift of the mediastinum to the lung which is obstructed if obstruction emphysema is the cause of the symptoms also the involved lung will appear smaller on inspiration than the normal lung On expiration the re verse happens and the mediastinum shifts toward the normal lung which is deflated whereas the affected lung still holds

some of the air The normal appears to become smaller and about the size of the obstructed lung This may be confirmed by fluoroscopy and x ray examination during the inspiratory and expiratory phase Where obstructive emphysema occurs the diaphragm of the involved lung appears to be raised

The following case is a typical example of an obstructive emphysema where there had been constriction of the bronchi in the left upper lobe due to an old tuberculous disease The *tuberculosis was arrested but due to constricture of the* bronchi leading into that lung there was an obstructive emphysema This patient was bronchoscoped and the fibrosis in the bronchi was reported but the wheezing cough and occasional dyspnea has been continuous since 1939 The only relief in such a case would be to remove the lung Many physicians had attempted to treat this patient for an allergic condition She was seen in 1952 and still has the same obstructive emphysema She still has pulmonary signs of an asthmatic condition with rales in both lungs and paroxysmal periods of dyspnea

Usually asthma is due to anaphylactic manifestations in sensitized persons which cause a constriction of the bronchi The anaphylactic *conditions in sensitized persons are the* most frequent causes of asthma The bronchi become contracted and produce a localized or generalized contraction of the bronchi throughout the lungs This form of asthma occurs in the early decades of life

We must assume that anaphylactic manifestations in sensitized persons are the chief causes for bronchial asthma and we must also assume that unless one finds the cause of the

production of this sensitivity, the asthmatic condition may persist for years

Asthmatic conditions occur frequently in persons who have a history of predisposition They have symptoms that may be traced to bronchial asthma and which have occurred in the father or mother or some member of the immediate family Their parents or some member of their family may have a history of hay fever, urticaria or a peculiar sensitivity to a food or odor One must rule out other conditions that may produce a similar picture Other conditions that should be looked for are cardiac and kidney disease

It has been stressed that in childhood and adult life up to the age of thirty there is probably an allergic cause unless we are aware of the asthma being due to the causes previously mentioned Asthma of the allergic form may occasionally occur past thirty years of age after forty years we should *assume that the cause is not allergic*

BACTERIAL ASTHMA

Bacterial asthma may be difficult to identify skin reactions are not easy to produce in humans But where there is no bacterial infection and no positive skin reaction the absence of the skin reaction does not rule out the condition There may be an associated polypoid sinusitis in asthma this should be looked for in the intrinsic causes of asthma

TREATMENT

Treatment of acute attacks are not difficult Common therapy is by injection of adrenalin chloride from 25 to 1 cc of 1000 solution of adrenalin chloride This may be repeated at

1/2 hour intervals until the attack is controlled Epinephrine as a vapor or aerosol may be inhaled in 1 to 1000 solution In severe asthmatic attacks oxygen is of great help One of the drugs to be used with caution *is morphine It is important* to remember that morphine may precipitate an asthmatic fatality When the patient is cyanosed there is nothing more helpful than oxygen and it is best given in an oxygen tent When severe attacks occur aminophylline is a very important drug In intravenous injections of aminophylline there have been a number of accidents When the same amount of aminophylline is put in a glass of tap water and given by rectum it usually is as effective as by the intravenous route in my experience Aminophylline should be administered in this way One of the precautions is to make sure there is a cleansing enema before giving aminophylline per rectum To repeat administration of aminophylline per rectum and maintaining the patient in an oxygen tent is the best method of treating status asthmaticus In severe cases of asthma ACTH injections have a definite value Cortisone is of great help in continuous asthmatic attacks When cortisone is preferred use small and continuous doses of ACTH about 20 mg two or three times a week

XXV PULMONARY EMBOLISM

Pulmonary embolism is generally considered to be a complication following a surgical procedure Its incidence as a post surgical complication varies from less than 1 0% in general surgery to about 2 5% in special forms of surgery We are more interested in the diagnosis in medical cases because while it is simple to make the diagnosis following surgery in medicine it is frequently overlooked and not diagnosed The complication in medicine may be as high as 13 0% In patients who have had long rest periods the incidence may be much higher and the diagnosis is made more frequently by the pathologist at the autopsy table than by the clinician during life

THE CLINICAL DIAGNOSIS

This may be very difficult the symptoms are most bizarre and there should be more educational time devoted to explaining these symptoms so that they can be more easily recognized We usually suspect such a diagnosis when there are symptoms and abnormal physical findings in a chest following surgery As a medical complication pulmonary embolism is too infrequently considered It is important that a diagnosis be made since pulmonary embolism usually recurs A recurrent attack may be fatal and could be pre

vented if the patient is treated properly Therefore though the clinical diagnosis is known to be most difficult the symptoms physical findings x rays of both lungs, electrocardiogram and other diagnostic tests of pleural fluid or sputa if present should be carefully considered When a diagnosis is suspicious it should be considered as equal to a positive diagnosis

Pulmonary emboli most frequently originate in thrombotic veins of the legs *This is the most common* cause but pulmonary emboli may also come from the pelvic veins during pregnancy puerperal sepsis prostatitis and mural thrombi of the right chamber of the heart Dislodgement of emboli from these sources is brought about by a rapid rise and fall of venous pressure incident to straining at defecation coitus lifting parturition or vigorous exercise this is called effort thrombosis

Pulmonary embolism may occur as a complication of any surgical procedure In general surgery the incidence varies and is considerably less than 1 0% When surgery is done in the pelvic area the incidence is increased and the figures are about the same in parturition The incidence of pulmonary embolism is high in amputation of the thigh

Pulmonary emboli are frequently associated with cardiac disease especially congestive heart failure coronary disease particularly following acute myocardial infarctions rheumatic heart disease especially with mitral stenosis and atrial fibrillation and hypertensive heart disease Prolonged immobility is often a cause of pulmonary embolism

Symptoms The symptoms of pulmonary embolism may easily be overlooked and as many clinicians state the actual

diagnosis may be very difficult to determine Pulmonary embolism once suspected should be treated as though a definite diagnosis had been made Treatment may prevent the recurrence of pulmonary embolism which could be terminal The knowledge or suspicion of small pulmonary emboli may prevent a fatal recurrence

Pleural Pain Sharp but momentary pain in one or both pleuritic areas may appear at either or both bases of the lung The pain in the pleura may be piercing and rapidly disappear Cough is a frequent occurrence and may disappear as rapidly as the pain in the pleura With the cough there may be a slight expectoration which may even be blood tinged and there may be an increase in the temperature, pulse and respiration rates These symptoms disappear rapidly and may not be mentioned to the physician Further attacks may occur and the symptoms again rapidly disappear When there is piercing pain over one or both pleura it is frequently referred to the corresponding shoulder Such fleeting attacks should make one suspicious of pulmonary embolism

Pain Over the Mediastinum This is common and suggests the possibility of cardiac attacks The mediastinal pains are very slight Pricking pain may suddenly appear over the pleura on one or both sides of the chest Attacks of mild fatigue and short episodes of dyspnea may also occur In more extensive pulmonary embolism the symptoms are more constant and simulate pulmonary diseases with cough hemoptysis dyspnea chills and fever Pleural effusions may occur and may be hemorrhagic One or both diaphragms

may be displaced and appear high in the roentgenogram or in the tomogram

Atelectasis may occur in both of the lungs and frequently resemble bronchial pneumonic patches Pain and tenderness over the chest wall may suggest pulmonary emboli The patient may become jaundiced With infarction of the left lower lung there may be pain over the upper abdomen

Case 1 A 40 year old male employed as a press operator The patient had pain in his right anterior thorax and close to the sternum and occasionally in the left anterior thorax He had occasional pain in his right occipital region Frequently the pain would last from five to six hours When the pain occurred it was very severe Occasionally he would be nauseous and was told he was jaundiced The pain frequently occurred anteriorly and close to the right mediastinum

An X ray dated 5 17 57 showed accentuation of the bronchial markings in the right root area and along the descending bronchi In his left lung the shadows were similar to those seen in the right lung There was a good deal of parenchymal calcification and the bronchi to the lower portion of his left lung were accentuated down to the diaphragm There were some small parenchymal infiltrations in the lower third of the left lung There were costo-phrenic adhesions in his right diaphragm and there was a similar condition in the left diaphragm with the costo phrenic angle also obliterated This patient exhibited symptoms of pain in his thorax—sharp and fleeting in character By the time he called his physician the symptoms were usually absent

Figure one was a reproduction of a roentgenogram taken



Fig 1

of his thorax and a similar condition was observed as in the x ray taken when he was first examined A tomogram was done and it was noted that the bronchi in both lungs were accentuated with calcification along the bronchi However in the left lung the accentuation was very marked and extended to the diaphragm Figure two was a tomograph taken at 8 cm in the anterior posterior position



Fig 2

In his history the patient complained of pain over his upper abdomen and stated he frequently was jaundiced After viewing the x rays a diagnosis of pulmonary emboli was suspected especially because of pain in the right and left thorax the right border of the mediastinum and the

rapid disappearance of all these symptoms The spasm of pain that occurred in the right upper abdomen can only be explained because of the peculiar pulmonary shadows that were noted in the lower portion of the left lower lobe Because of the bizarre symptoms that occurred in the thorax and mediastinum an electrocardiogram was taken and showed that he had a prolonged S in Lead 1 and a slurred QRS in Lead 3 This occurrence suggested the possibility of a right bundle branch block The definite diagnosis of such a block could not be confirmed because there was no similar slurring in 2 or 3

It is to be noted that in Case 1 the patient showed no abnormal physical findings in his lungs and over his heart There was a bizarre clinical condition where pain occurred over the mediastinum and abdomen and there were also repeated attacks of jaundice The patient was treated with anti coagulation therapy and the varicosities in the right and left legs were removed

Case 2 A 55 year old female who is an instructor of rehabilitation therapy The patient stated that three months previously she developed a cold in her head which lasted for about two and one half months She finally went to a physician who fluoroscoped her and told her she had a pneumonitis She was advised to be hospitalized

Her symptoms were chiefly pain over the right thorax and over the mediastinum She had also been jaundiced about one month previously There was a question as to whether she might have a neoplastic disease of the right lung

Figure 3 is a reproduction of a conventional X ray taken when the patient was first seen on May 31 1957 it showed a disseminated infiltration in her right lung that extended



Fig 3

down from the seventh rib to the pleura The bronchi down from the root region to the mediastinum were accentuated There was also some pathology noted in her left lung about the vicinity of the seventh rib The root shadows in the left hilar region showed some evidence of calcification The physician who thought she had pneumonitis also thought she might have a neoplastic disease and a tomographic examination of the patient was made a sputum was sent to the Papanicolaou Laboratory for examination for tumor cells The x ray of May 31 1957 showed a bizarre type of bronchopneumonia in the lower third of the right lung

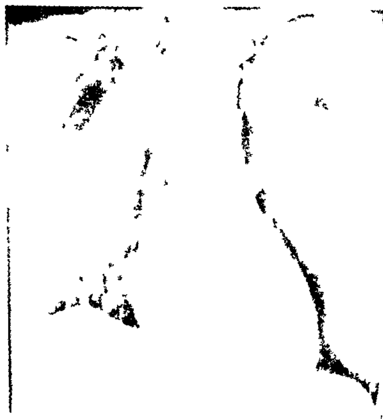


Fig 4

Tomographic study was done Figure 4 is a reproduction of the 4 cm anterior posterior x ray which revealed a shadow to the right of the spine that extended from the sixth rib down to the diaphragm It is noted here that the right diaphragm is higher than the left diaphragm There is an adhesion from the inner third of the diaphragm to the pleura and the pleura seems to be obliterated There is con

siderable calcification and extensive shadows are seen to the right of the spine Tomographic studies of the lung were taken from 4 cm to 14 cm The 4 cm film has been previously described the 6 cm film showed the costo phrenic angle to be obliterated on the right The peculiar string like adhesion to the right pleura had disappeared and the same shadow that was described in the roentgenogram at 4 cm had disappeared The right diaphragm was raised Calcification was still present near the root region The left lung and bronchial markings to the left lower lung seem to be more accentuated and reach the left diaphragm the 8 cm A P film showed the right diaphragm to be raised and the shadow noted before had disappeared Bronchial markings along the left lower bronchi were still accentuated the 10 cm film showed some resolution of the pathology previously seen but accentuation of the bronchi to the right and left hilar regions was still seen the 12 cm A P film showed a new shadow in the right lung which extended to the pleura and descended down to the diaphragm and over to the mediastinum This shadow was very anterior in the lung and seemed to be in the middle lobe The right costo phrenic angle was still obscured The bronchi had become less exaggerated Figure 5 is a reproduction of the 14 cm film in the anterior posterior position this level is in the very anterior portion of the lung and shows that the shadow previous described had become very exaggerated extending from the mediastinum straight to the pleura The costo phrenic angle was still obliterated In this tomographic series the right diaphragm always seemed to be distinctly higher than the left especially in the 8 cm film where it was considerably higher

The patient was bronchoscoped and the examination re



Fig 5

vealed no abnormal findings in the bronchial tree the biopsy was negative for carcinoma cells anti coagulation therapy was begun

Anti coagulation therapy was continued for six months When this patient had an electrocardiogram done on 5 31 57

she had a left axis deviation and an old posterior infarct and an accentuated Q3 Here is a severe case that originally suggested a broncho pneumonia and x ray examination revealed a shadow that could possibly be a neoplasm but which finally completely resolved A possible source of the pulmonary emboli was looked for and both legs were examined In the left leg the veins were normal in the right popliteal space there were many small veins visible and there were small varicosities on the periphery of the right thigh

Tenderness was present over the upper thorax, the lower thorax and close to the mediastinum The position of both diaphragms was demonstrated in X rays of the lungs and also in laminographic studies of the lungs The anterior films of the tomogram demonstrated an apparently normal diaphragm but in the posterior films the diaphragm appeared raised

Case 3 A 68 year old white female This patient had been ill for the past ten weeks She had developed a productive cough which was very severe She had precordial pain which rapidly disappeared and also had severe left scapular pain She had many upper respiratory infections accompanied by productive cough in the past few years The present attack was very similar to those she had in the previous few years

She was seen with a diagnosis of a possible carcinoma in the left upper lobe with some abscess formation in the mid portion of the lung When examined there were many rales over both upper lobes both anteriorly and posteriorly Associated with these sibilant rales were heard many moist rales The heart was normal except for the fact that over the apex of the heart a systolic sound transmitted towards

the axilla was heard and there was an accentuation of the second aortic sound

In view of the fact that there was a question about neoplastic disease a Papanicolaou test was done on her sputum which was reported negative A sputum was also examined for tubercle bacilli and was also reported negative An electrocardiogram was taken 7 15 57 and revealed a normal tracing

A sensitivity test for antibiotics showed the patient to be sensitive to achromycin panmycin terramycin albamycin neomycin and chloromycetin She was found resistant to the following albamycin aureomycin erythromycin matromycin cathomycin dihydrostreptomycin furacin and furadantin It was decided to put this patient on a sensitive antibiotic for one month to be changed monthly to another sensitive antibiotic She was given sigmamycin achromycin panmycin and chloromycetin for one month each the antibiotics to be given only for one month so that the patient does not become resistant This patient did very well with her bronchial condition on this regime When she first became ill she had a large mass in the left upper lobe and there was evidence of some shadows over the left hilus that showed multiple cavitation She had many sibilant moist rales over the left upper lobe both anteriorly and posteriorly

Figure 6 is a reproduction of a posterior anterior film of the chest taken when this patient was first seen on 7 15 57 This x ray demonstrated that she had involvement of the parenchyma of the left upper lobe with considerable accentuation of the bronchi leading to the hilus She had an interlobar thickening and in the inner third of this area one could see multi loculated areas of rarefaction as though there

were multiple little cavities present In the right lung there was evidence of a good deal of calcification along the bronchi and the root area extending almost to the diaphragm A diagnosis of pulmonary infarction was made in both the left

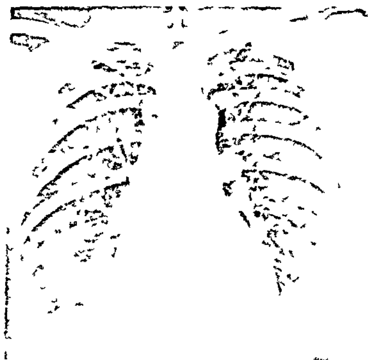


Fig 6

and right lung probably more severe in the left, complicated by a severe bronchial infection There was no odor to the sputum

Figure 7 is a reproduction of a posterior anterior x ray of this patient's chest taken 11 16 57 The shadows noted in the left upper lobe had resolved At the hilar areas there

was accentuation of the bronchial markings where there was some calcification The interlobar shadow that had been noted in the left lung had almost completely disappeared and the area of infiltration below it with rarefaction had disappeared The bronchial markings previously noted were not



Fig 7

as severe The shadows in the right lung were much improved Physical examination at this time revealed a complete disappearance of all physical findings The patient felt very much improved Her cough subsided and she could not raise any more expectoration Her mediastinal and shoulder pains had disappeared She had gained eleven

pounds and she was completely free of her symptoms This patient was not treated with anti coagulant therapy because of her age which was 68 years

Pathology The recognition of pulmonary embolism really dates back to 1822 when Lainec described it Pulmonary embolism may occur with or without infarction In small emboli whether infarction occurs or not is still controversial It is important that the pathologist record the fatal cases there is very scant literature on such lethal pulmonary emboli since they exist only for a short duration and as a rule do not come to the autopsy table When necropsy is done in massive cases the mediastinum and heart are found to be pulled over to the affected lung the diaphragm is usually raised and the lung may even be collapsed firm and airless The airless lung will sink in water Bilateral involvement of the lungs is a frequent occurrence

One finding common in infarcts of the lungs is the close proximity of the lesion to the sub pleural area The infarct is usually firm and varies in color from red to black The area involved also varies it may be as small as a pea and often a whole lobe or even the entire lung is involved Characteristically the involved area is usually in close contact with the pleura and the lower lobes are a favored site The areas are usually dark in color when they are recent and when they are much older they are reddish in color and appear like diffuse blood clots

Infarcts may be complicated by pneumothorax or hemopneumothorax or even a pyo pneumothorax The microscopic examination shows the air cells and walls distended with red blood corpuscles In very massive cases it is not unlikely to

find an atelectatic lung a high diaphragm in the side involved and also the mediastinum pulled toward the side involved

DIAGNOSIS

There may be great difficulty in making a diagnosis of pulmonary emboli the findings may be interpreted as a broncho pneumonia an atelectasis pleurisy pericarditis myocardial infarction spontaneous pneumothorax and congestive heart failure when the latter might be the source of the pulmonary emboli Where there is evidence of peripheral venous thrombosis pulmonary embolism should be considered Where there is sudden onset of pleuritic pain with no obvious explanation of this condition a broncho pneumonia which does not follow the usual course a sudden pleural effusion or a sudden puzzling post operative fever a diagnosis of pulmonary emboli should be entertained

Pulmonary embolism frequently follows congestion of the lungs in heart disease Hemoptysis may or may not occur but in the presence of the mitral stenosis one should think of the possibility of pulmonary embolism

Electrocardiograms The electrocardiogram may reveal nothing or may help to diagnose pulmonary emboli The following patterns may occur

Pattern 1) Lead 1—Prolonged S
Lead 3—Accentuated Q
Pattern 2) Lead 1—Prolonged S
Lead 3—Accentuated Q_3
Inversion of T_3

XXVI ACUTE LOBAR PNEUMONIA

Lobar pneumona is a pneumococcal infection of the alveolar spaces of the pulmonary tissue and one or more lobes may be involved When the disease is acute it appears suddenly and *the characteristic symptoms are sharp pain in the chest* chills and high temperature It is usually associated with an extensive consolidation of part of one or both lungs there may be evidence of intoxication and the progress of the dis ease *is unpredictable It may go on to a fatal ending* or to a resolution and disappearance of most of the symptoms There may be sudden disappearance of all symptoms as in the crisis and the patient rapidly appears well On the other hand *symptoms may disappear slowly and recovery* be more gradual this occurs in lysis where the recovery is due to slow absorption of the infection When there is continuous involvement of the alveolar air cells the concept of broncho pneumonia is applied to the disease When this occurs the disease may terminate either by crisis or lysis

Ninety per cent of lobar pneumonia are caused by pneu mococcus In patchy types of pneumonia and broncho pneumonia there may be other organisms such as strepto coccus hemolyticus Friedlander s bacillus staphylococcus aureus hemophilus influenza and even tubercle bacilli The

organisms should be identified usually pneumococcus is the most prevalent organism but one should also look for other possible organisms If the pneumococcus is found it should be typed this can be done either from the sputum or the blood stream Streptococci hemolyticus are usually found in about two to three per cent of all cases Friedlander s bacilli is one to three percent staphylococcus hemophilus influenza and tubercle bacilli occur in this order of frequency Streptococcus may be a primary infection and may follow a tonsillitis pharyngitis or scarlatiniform rash Frequently streptococcus infections may be a complication of influenza infections When the disease is very diffuse through both lungs Friedlander s bacillus should be suspected especially where there is profuse bilateral infection of the lungs In Friedlander s bacillus infections the sputum may be viscid and sometimes cherry colored These organisms can easily be demonstrated they have gram negative stain and are readily identified Blood culture may be positive

In atypical pneumonias a search for the following organism should be made It is not uncommon to find psittacosis this disease should be suspected in all cases where birds inhabit the household for instance parrots parakeets etc

It is common to divide lobar pneumonia into three stages the first stage is inflammatory the second stage is a filling of the alveolar cells or red hepatization the third stage is that of gray hepatization In all three stages the lung appears to be firm

The first stage is rarely seen at autopsy in this stage the capillaries of the alveolar walls are diluted with plasma There is an outpouring of protein rich edematous fluid into the alveolar spaces thus becomes a culture media for pneu

mococci These organisms are frequently transported from one alveolus to another from lobule to lobule and lobule to lobe The first stage is called a stage of engorgement

The second stage is the most common stage found at autopsy here the lung is usually found in red hepatization At this stage there is an inflammatory exudate filling all the alveolar spaces and the lobe appears firm and dry The alveoli appear to be filled with a coarse mask of fibrin in which there are many pneumococci red corpuscles polymorpho nuclear leukocytes and desquamated epithelial cells There also may be some mononuclear wandering cells or lympho cytes The leukocytes are clearly outlined and turgid Many of these leukocytes show active phagocytosis and contain many pneumococci The lobes involved may be one or sev eral they are usually hard and heavy The hardness is caused by consolidation of the lobes which appear to be airless and easily sink in water

In the third stage which is that of gray hepatization the red cells have undergone laking and seem to have disinte grated When the leukocytes are noted here they appear to be partly ragged Many desquamated epithelial cells are present Pneumococci when noted usually do not stain bril liantly as in the previous stage of red hepatization In this stage the blood has difficulty passing through the lung and the latter appears to be avascular This is when crisis usually starts It has been noted that patients rarely die during gray hepatization When symptoms do not stop abruptly lysis may occur this process is usually explained by pneu monic areas becoming avascular more slowly In the first two stages of pneumonia there is an absorption of the toxic substances in the third stage the symptoms cease

Symptoms When first seen there may be a minor history of illness Usually there is a history of a mild pharyngitis before the present symptoms occur This nasopharyngitis is frequently followed by chills which last from a few minutes to almost one half hour There may be shaking chills these may be so severe that the bed shakes There is marked chattering of the teeth the chills usually occur at the beginning of the pneumonia There may be marked chest pain this occurs at the very onset of the pneumonia and may even precede the chills Pain is very characteristic of pneumonia it may be a stabbing pain and is often accompanied by cough and rapid respiration The pain may be caused by an inflammation of the pleura over the site of the pneumonic area The cough is frequently associated with a rusty sputum This is due to the mixture of mucus and blood formed in the inflammatory process in the alveolar spaces in the lung Fever and toxemia are constant with the pneumococcal process The patient may complain of severe weakness in the legs and this is associated with marked prostration When the fever and toxemia cease gray hepatization occurs

Physical Findings Pneumonia should be suspected where there is any diminution in the intensity of sounds in the early stages Frequently this may be the only finding of superficial inflammation of the lungs in early inflammation of the central portion of the lung there may be no abnormal breath sounds Very often because the pathology is centrally located the superficial portion of the lung may be emphysematous and appear normal When breath sounds are diminished suspect the occurrence of a pneumonic process when lobes are consolidated there is a marked diminution

of breath sounds and bronchial breathing may be heard over the lung When symptoms persist and dullness increases particularly over the pleura search for a probable pleural effusion which can easily be verified by inserting an aspirating needle over the suspected area When a pleural effusion occurs there may be either a simple effusion or an empyema

DIAGNOSIS

Whenever an acute febrile disease occurs with pain in the chest and expectoration of viscid sputum often blood tinged in appearance a lobar pneumonia is suspected In all such cases, examine the sputum or blood stream for pneumococci When signs of consolidation of the lung appear the diagnosis is confirmed The bacteriological diagnosis can be determined by sputa cultures The findings of these pneumococci or mixed organisms should be of great assistance in planning therapy

TREATMENT

Penicillin is still the best therapy for the treatment of pneumococcal pneumonia The various strains of pneumococci are very sensitive to penicillin and they are inhibited in blood cultures in a concentration of 0 01 units per millimeter A large initial dose of one million units is advisable and treatment should be maintained until the temperature remains normal for three days The penicillin may be given intramuscularly or orally In oral dosage give at least five times the dosage given intramuscularly The bacteria present usually clears in twelve hours and this is followed by defervescence and a striking disappearance of symptoms When penicillin does not relieve the fever and toxemia of

lobar pneumonia in forty eight hours complications may occur In pulmonary infarction where the alveolar spaces may be filled with blood and partial atelectasis occur a secondary infection may arise it is not uncommon to see massive pulmonary disease of either lobar pneumonia or broncho pneumonia disappear with therapy without the pulmonary embolism or infarction being suspected

Oxygenation Patients with pneumonia may become cyanotic and oxygen should be given for relief of both the cyanosis and the frequent occurrence of cough Oxygen may be given in a tent mask or a nasal catheter When a tent is used eight to ten liters of oxygen per minute should be supplied When a mask is used pure oxygen may be used because the contained expirations supply the necessary carbon dioxide When a nasal catheter is used the mixture should consist of 95% oxygen and 5% CO and in this way a sufficient amount of carbon dioxide is inhaled Never use a catheter with 100% oxygen

XXVII FOREIGN BODY ASPIRATIONS

Foreign bodies are frequently aspirated into the tracheo bronchial tree in children and adults The aspiration is frequently overlooked because the history which is so important is often forgotten and not told to the physician The occurrence of symptoms of spasm of choking gagging coughing irritation of the pharynx and wheezing suggest the possibility of the aspiration of a foreign body into the tracheo bronchial tree

Asthmatic Symptoms These symptoms often increase or subside as the foreign body is deeply aspirated in the lung and changes its position Symptoms and physical signs in the chest after aspiration will depend on the position of the foreign body in the bronchial tree Asthmatic symptoms are frequently associated with aspiration of a small foreign body and the first diagnosis is that of asthma

A patient with symptoms of asthma, had wheezes over the right lung on physical examination X ray of the chest revealed a great deal of calcification in the right hilar region and gave the appearance of there being some small pulmonary stones in this area Tuberculosis was suspected and the sputum was carefully examined it was negative for

tubercle bacilli The diagnosis of tuberculous disease was discredited One morning while she was being examined she spat up some pulmonary stones and one of these was examined for tubercle bacilli surprisingly it revealed the presence of tubercle bacilli The patient was placed on isoniazid PAS and streptomycin therapy With this treatment she improved but still had asthmatic attacks and still expectorated small pneumoliths Apparently some of these small stones were aspirated into the small bronchioles causing an obstructive emphysema and asthma After six months of therapy the patient had improved considerably her asthmatic attacks disappeared and no more pneumoliths were expectorated This patient frequently expectorated small stones which were aspirated into the bronchial tree when this occurred it caused a mild obstructive emphysema with asthmatic signs and a good deal of wheezing in the upper portion of the right lung At times when she had an obstruction of the small bronchi there was absence of breath sounds over the area of the lung in which the stones were aspirated The patient made a complete recovery

Many foreign bodies are not opaque in x rays Foreign bodies may be aspirated by children and if they obstruct the bronchus may cause an atelectasis Aspiration of foreign bodies is a frequent occurrence in children and the presence of a foreign body may be determined by a shifting of the mediastinum to the side of the lung involved This is frequently produced by the presence of a foreign body such as a peanut lodging in a bronchus and causing an obstructive emphysema

The same obstruction can be caused by a tuberculous growth of the bronchus or a tumor in the lung Obstructive

emphysema can be seen in fluoroscopy and checked by x ray taken on inspiration and expiration In such obstructions bronchoscopic examination will reveal the foreign body or show the presence of a tumor or tuberculous disease causing obstruction of the bronchus Foreign bodies that are lodged in any part of the tracheo bronchial tree can be removed by bronchoscopy The x ray will disclose a foreign body obstruction or if it is not radio opaque the obstruction may frequently be seen by the shifting of the mediastinum away from this obstruction during expiration

This should also be kept in mind with non opaque foreign bodies entering the tracheo bronchial tree and the symptoms and findings depend chiefly upon the area where the foreign body lodges Take for example the aspiration of fish bones the important thing here is the history If a patient develops a cough following such a history a bronchoscopy is important There may be inflammation of the lung surrounding the bronchus in which the fish bones lodge and should the signs and symptoms not disappear bronchoscopy must be done as soon as possible

It is important to remember that many cases are greatly complicated because there is no history of aspiration of a foreign body A good example of such a case is that of a man who was admitted to a hospital with a lung abscess without a history of aspirating a foreign body The lung abscess was bronchoscoped and a foreign body was found resembling the stem of a clay pipe This man s parents then recalled that as a youth the patient was riding a bicycle and was struck by an automobile and knocked unconscious The patient never knew he had swallowed the tip of the pipe which he

had in his mouth at the time of the accident Apparently he had no knowledge of it until he developed a lung abscess The removal of the tip of the clay pipe completely cured his pulmonary disease

Aspiration of food may cause a lung abscess In fact any foreign body or any solid material aspirated with the knowledge of the patient may cause such an abscess It is not uncommon for a child to swallow a loose tooth with no knowledge of having done so The first symptoms are those resembling a cold in the thorax After carefully determining the diagnosis bronchoscopy should be done and the tooth removed All suspicious cases should be x rayed in inspiration and expiration and if negative a bronchoscopy should be done to be certain about the diagnosis

Children frequently place small articles in their mouths we have already spoken of nuts peanut kernels and nuts combined with candy which could easily be aspirated especially when thrown into the mouth Pieces of candy can be aspirated as well as cooked beans fragments of string beans carrots and even mashed potatoes It is well for parents to caution children not to speak with food in their mouths Accidental aspiration of metallic objects is common particularly tacks or pins which have been held in the mouth dental fillings or a broken tooth There is constant danger of small fish or meat bones being aspirated into the trachea and lodging in the bronchi When a large particle of meat is aspirated especially in adults there may be immediate signs of occlusion of the trachea with signs of asphyxia which may rapidly be fatal In these cases cyanosis may suddenly appear and a tracheotomy may be indi

cated immediately Shifting and manipulation of foreign bodies in the trachea is dangerous and bronchoscopy is a safer method of removing foreign bodies

Early Symptoms The early symptoms of any aspiration of foreign bodies in children and adults are spasms of choking gagging coughing and irritation of the thorax and tempera ture These symptoms may disappear and a pneumonic process may develop or a lung abscess When the early symptoms are recalled a simple bronchoscopy which is not dangerous may rule out the possibility of any foreign body When wheezing occurs it should be suspected that there may be a small obstruction of the bronchi the simplest way of ruling out such a condition would be bronchoscopy and roentgenograms of the lungs to rule out the obstructive emphysema If the bronchus is actually obstructed there would be no shifting of the mediastinum if there is a partial obstruction the mediastinum will shift toward the side ob structed When the mediastinum is adherent it will not shift In the check valve variety of obstruction of the bronchi there is little change in the passage of air on inspiration but during expiration the air is trapped and the obstruction emphysema is produced Breath sounds may be diminished over the whole thorax and even be transmitted to the oppo site side On fluoroscopy there may be slight difference between the two lungs on inspiration On expiration the air is trapped where the block occurs and there is less change than where the lung is not obstructed In the normal lung during expiration there is less air and it therefore appears smaller than the involved lung The diaphragm on the obstructed side raises normally

TREATMENT

Whenever a diagnosis is made of a foreign body it should be removed and usually this can be done by bronchoscopy In the by passed type the physical findings are scant A few rales over the foreign body and occasional wheezes are heard in this type In the check valve variety the breath sounds are diminished or absent over the emphysematous area rales and wheezing are heard over the area of the check valve and are transmitted to the chest wall in both lungs Where a complete obstruction of a bronchus occurs there is absence of breath sounds and the mediastinum and surrounding structures are pulled to the side of the obstruction usually the diaphragm is raised on the side of the obstruction Treatment of complete obstruction by foreign body is removal by bronchoscopy after this is done the lung completely re expands

Wherever bronchial emphysema exists and persists bronchoscopy should be attempted Where there is a complete obstruction of bronchus the first thought should be that of neoplasm Other diseases may produce a similar condition For example a tuberculosis can block a bronchus or a gland can press on a bronchus and produce an atelectasis

XXVIII CRUSHING INJURIES OF THE CHEST AND TRAUMATIC ASPHYXIA

Crushing injuries of the chest are common in military medicine but they also may happen in civilian practice Accidents which produce great force on the thorax may result in direct injury to the chest and complications may be present at the same time Automobile accidents may result in multiple injuries involving the skull the thorax the abdominal cavity and the lower extremities the total damage must be ascertained as soon as possible if life is to be saved Crushing injuries are chiefly penetrating and explosive when incurred during warfare however the same may be true of those incurred during civilian life In cases of this type the chest physician should ascertain the injury to the thorax and should seek help in ascertaining the amount of injury to the other organs

Hypoxia is usually the chief offender after a crushing injury to the thorax and follows disturbance of the delicately balanced cardio respiratory function Respiratory failure may be produced by mechanical failure of the respiratory function acute pulmonary edema hemorrhage and even cardiac tamponade may occur When respiratory failure occurs one should look for paradoxial breathing where an air flow exists between the crushed lung and the good lung there may be

an obstruction of the airway by blood Here one should determine the source of the blood which causes obstruction of the airway and search for mediastinal emphysema Gastric dilatation may cause the respiratory failure especially where there is an elevation of the diaphragm A bilateral tension pneumothorax may be present

SYMPTOMS

In a crushed thorax the following should be promptly determined

1 Flapping of the chest
2 Loss of blood and the site of hemorrhage
3 Whether pleural fluid exists in either pleural cavity
4 Acute pulmonary edema
5 Compression of the airways

The chest can be disturbed by pulmonary concussion or lung blast The force of an explosion may cause immediate death In explosive accidents there is profound prostration associated with marked dyspnea tachycardia or pain in the chest Cough is a frequent occurrence and hemoptysis is usually present The most important symptom is bulging of the chest and there may be mottled areas of density seen in the x rays of the chest It should be remembered that blasting concussion may also produce chest injuries Crushing injury frequently occurs where walls are demolished and the victim may be pinned down in the debris of a collapsed house falls from great heights often result in great trauma with multiple fractures of the ribs It is surprising that it is possible to sustain life in some of the more severe injuries of this type

TREATMENT

In these conditions all measures to sustain life must be given promptly If there is hemorrhage determine where it is coming from It may come from the direct injury to the chest wall In order to stop bleeding of the lung collapse the lung by pneumothorax Watch for intercostal vessels that may be injured and actually bleed into the open lung space Shock should be treated Consider the trio of symptoms—shock dyspnea and pallor—which should arouse suspicion of a crushing injury to the chest When blood obstructs the airways an attempt should be made to remove the blood this frequently can be done by passing a catheter through the nose into the bronchus and aspirating by suction the blood debris in the airways Blood loss should be replaced Usually citrated blood is given from two to three liters by transfusion Saline should never be used as it may produce pulmonary edema

Pneumonia This should be anticipated and the thorax aspirated In tension pneumothorax drainage is frequently relieved by introducing a catheter into the intercostal area and draining under a few cc of water until the pneumothorax is relieved It is wise to wait a day or two to allow the pulmonary vessels to thrombose Oxygen is an important part of the therapy Use an oxygen tent and give ten liters of oxygen per minute The oxygen administration can be discontinued once the patient is safe

Flapping Chest It is important to control a flapping chest This can be relieved by adhesive strapping by encircling the

entire thorax Usually strapping the chest and administration of sufficient oxygen therapy overcome noxious factors

Complications When the bleeding stops aspirate the pneumothorax or pneumomediastinum If suction of the hemothorax or mediastinal cavity is not successful surgery should be resorted to

If cardio tamponade produces pathology in the lungs this *condition may be controlled with a pneumothorax The fol* lowing suggestions are helpful

1. In a crushed lung pulmonary embolism is a frequent complication
2. When a hematoma is acquired in the area of the upper sternal notch the site of incision should be covered with petroleum gauze This stops blood and air from entering into the airways
3. A duodenal tube will frequently aid gastric dilation
4. A large herniation of the abdominal viscera may occur through a ruptured diaphragm
5. Should a crushed chest not be controlled by strapping the thorax with adhesive tracheotomy frequently helps this condition
6. Bronchoscopy will prevent the accumulation of tracheo bronchial secretions which may lead to both atelectasis and pneumonia

An injury to the chest may appear very severe and the prognosis appear very poor but the patient frequently survives In crushed conditions of the chest hypoxia must be corrected Place the patient under complete oxygenation To be sure the patient has sufficient oxygen have at least ten

liters or more of oxygen to a tent The patient is generally too ill to be x rayed but it should be determined if he has any changes in his pleura by percussion and auscultation if there is a question a needle may be inserted to find blood or air which can be aspirated The airways should be examined carefully to see if they are blocked Obstruction in the air ways may be aspirated by simply inserting a catheter into the nose and into the bronchus Tamponade should be deter mined and an attempt made to relieve it by aspiration Fre quently a simple ligature of an intercostal artery can be of great help There should be some delay before doing any surgical procedures When the patient becomes more com fortable a complete estimate of the damage done to the ribs and organs should be made and this should be corrected surgically later For example there should be no rapid attempt to correct a post traumatic hernia

TRAUMATIC ASPHYXIA

Traumatic asphyxia may be caused by major accidents like the collapse of a building or in cave ins of excavations Pa tients may be crushed by two moving objects such as auto mobiles or pinned under overturned automobiles or crushed in elevator accidents This asphyxia has already been de scribed in the section on crushing injuries to the chest There are many ways asphyxia may be caused and treated Some of the chief causes are obstruction of the trachea by aspira tion of blood especially from an injury of a blood vessel in the thoracic wall an injury to the chest itself an injury to the pleura or an injury to the blood vessels in these areas or to the intercostal areas with bleeding into the lungs the pleura or mediastinum

Symptoms Persons who are crushed look weird they have deep purple discoloration of the face the neck or even as far down as the clavicles The coloration looks fantastic this may disappear in one or two weeks Marked discoloration is due to blood in the dilated capillaries of the skin

Emphysema of the subcutaneous tissues may occur over the face the trunk thighs or even the fingers or toes When the lung is lacerated and the parietal and visceral pleura of the lung is torn air may escape into the pleural cavity during inspiration and cause a partial pneumothorax On expiration the rent may collapse with increased intrapleural pressure air is forced back through the visceral pleura into the subcutaneous tissue and may spread over the entire body This may be ameliorated in some measure by decreasing intra pleural pressure and when the patient is more comfortable an attempt should be made to treat the visceral pleural and the parietal pleural lacerations A fixed aspiration pneumothorax under water may reduce the pressure in the pneumothorax

Hemothorax Differentiation of a simple hemothorax from a hemo pneumothorax is important In a simple hemothorax the mediastinum is pushed to the opposite side and the fluid level of the hemothorax is curved to the height of the axilla A hemothorax may arise from a fractured rib which does not tear the parietal pleura Bleeding may be stopped by a ligature passed around the affected rib this frequently stops future bleeding A search for intercostal arterial bleeding should be made in these cases When the bleeding is controlled the hemothorax should be aspirated In severe hemothorax blood escapes into the pleural cavity and the fluid

level is straight instead of curved Laceration of the inter costal vessels and laceration of the lung may occur in such conditions In these cases entrapped air may reach enormous pressures and should be relieved by aspiration or continuous aspiration under water When bleeding has been controlled entrapped blood in the pleural cavity may be removed and the pneumothorax may be continued

XXIX MISCELLANEOUS ACUTE PULMONARY SYNDROMES

NORTH AMERICAN BLASTOMYCOSIS

North American blastomycosis is frequently called Gilchrist s disease The disease is caused by a yeast like fungus which may appear in cutaneous pulmonary bone or systemic form or in different areas at different periods The etiological factor is always the same The fungus infection is susceptible to ether which may be given in olive oil by rectum A patient who had involvement of the skin close to the right elbow and the bones of the left ankle along with enlarged glands at the right elbow and a miliary involvement of both lungs recovered from blastomycosis by having ether poured over the skin involved and into the surgical wound over the left ankle and the administration of ether one ounce in four ounces of olive oil by rectum The patient made a complete recovery from the disease The diagnosis may be established by identifying the fungus in pus sputum or tissue specimens The organism appears as a thick well rounded body it occurs singly in pairs and budding forms are common Use Sabouraud s medium at room temperature or blood agar at 37° C

COCCIDIOIDOMYCOSIS

This disease may be an acute infection which usually has a very good prognosis It may develop into a progressive coccidioidomycosis When it does it may produce a generalized granulomatous disease with a high mortality instead of a good prognosis The disease may be found in almost any part of the country but most commonly in Southern California It is wise to suspect this disease in any person that has lived in Southern California

The cause is the *coccidioides immitis* which multiplies in the tissues by endo sporealation and the organisms are spherules which have a thick hyaline capsule from about 10 to 20 micromes in diameter containing 10 to 200 endospors When infected material from tissues sputum or other discharges are planted on Sabouraud s media the endospors germinate and grow mycelia Skin sensitivities to coccidioidin is a permanent sensitivity in contrast to the presence of precipitins and complement fixing antibodies which slowly disappear after recovery It is known that with a positive skin reaction coccidioidomycosis infection produces the symptoms *that* parallel pulmonary tuberculosis A negative tuberculin reaction and a positive skin reaction to coccidioidin would suggest a diagnosis of coccidioidomycosis *Patients* may *have a* primary infection which usually do well Occasionally patients may develop multiple areas of infection throughout the lung In spite of these extensive lesions they *do fairly* well and may exist for a long period of time

There is no definite treatment for coccidioidomycosis that we know *Some of these cases have been treated* with ether in oil by rectum during the acute periods As a rule the gen

eralized types are very difficult to treat particularly where there are many cavities and the development of cysts Removal of some of these cysts by surgery has been helpful in certain cases

HISTOPLASMOSIS

This disease is an infection which is caused by *histoplasma capsulatum* The infection usually is caused by inhalation of dust which contains many spores Epidemics have occurred following inhalation of dried dead pigeons chickens and even bat dung The disease is diagnosed chiefly in x rays that appear to be old tuberculous pathology The disease frequently resembles tuberculous infection Histoplasmosis may involve a great many areas in the body the pulmonary the lymphatics the liver and the spleen Often the diagnosis is made chiefly because of its close resemblance to tuberculosis at times when the sputum is negative an attempt is made to differentiate histoplasmosis from tuberculosis

Definitive diagnosis is either by biopsy or by positive skin reactions to histoplasma Skin tests are performed when one is unable to diagnose tuberculosis in the lungs or in the glands

It is important to realize that the disease may progress for a long time without causing symptoms There are times however when histoplasmosis may cause large areas of disease in one or both lungs with acute symptoms Here a skin test with 0 1 cc of a 1 to 1000 of a standardized histoplasma solution should be made If after 48 to 72 hours a skin reaction appears similar to that of a positive tuberculin reaction a diagnosis of histoplasmosis is warranted

LOEFFLER'S SYNDROME

This is a type of pneumonitis characterized by transient and migratory eosinophilic infiltrations of the lung. In such patients there is usually a great increase of eosinophiles in both blood and sputum. This type of pneumonic infiltration frequently occurs in individuals with suggestive histories of either urticaria or asthma; the disease usually occurs in young adults. There is a very bizarre pulmonary picture. The disease is widespread and usually bilateral consisting of small areas of pneumonic infiltration. The small areas of pneumonic infiltration may be transitory and it is not uncommon for *these shadows to be seen in the roentgenograms of the lungs* and then to disappear rapidly. Both the sputum or the blood may show numerous eosinophiles. The pneumonic shadows may be due to parasitic worms like ascaris. Ascaris infestation may produce a pneumonitis with eosinophilia. Bronchial asthma associated with pneumonitis may produce the typical picture of Loeffler's Syndrome. The disease usually runs a limited and benign course. The diagnosis is chiefly made by the finding of rapidly developing shadows in the x rays of the lungs which disappear with equal rapidity. There is also a rapid appearance of the eosinophiles in both the sputum and blood.

ACUTE FUNGUS INFECTIONS OF THE LUNG

Fungus infections are frequently the cause of infections of the lung and they may either be the yeast or yeast mold type *Candida albicans* frequently called *monilia* is a yeast like type of infection. Blastomycosis, coccidioidomycosis and histoplasmosis should be differentiated.

CANDIDA ALBICANS

Moniliasis is caused by a yeast like fungus Candida Albicans This fungus is frequently found in the sputum it causes a bronchitis and a possible pneumonia The patients are not very toxic the prognosis is favorable even when candida albicans can be found in the sputum in large quantities

TREATMENT

The employment of one ounce of ether in four ounces of olive oil by rectum the ether is eliminated through the lungs and destroys the candida albicans

PARAFFINOMA OR OIL PNEUMONIAS

Very frequently when castor oil or nasal oils were commonly used paraffinoma or oil pneumonias were seen We found considerable deposits of these oils at the base of the lungs The treatment for these cases is simple Postural drainage over a period of time eliminates these oil deposits

Castor oil frequently produces similar paraffinoma The shadows seen in the roentgenograms suggests an early carcinoma With proper therapy and postural drainage these shadows rapidly disappear

Part IV

DIABETIC EMERGENCIES

HOWARD F ROOT M D F A C P D I M

XXX | DIABETIC EMERGENCIES

General Considerations Any emergency medical or surgical may occur in a diabetic patient and thereby assume unusual features or characteristics dependent upon the patient s diabetic status Under some conditions the diabetes may render the problem medical or surgical much more difficult from the diagnostic and therapeutic points of view If the diabetes is mild of short duration and under adequate control no difficult problem may be superimposed upon the emergency situation On the other hand if the diabetes is of long duration without adequate dietary treatment or skillful use of insulin and without continuous medical supervision then the patient may present a lowered resistance to infection poor healing ability consequent upon malnutrition or even diabetic acidosis States of diminished consciousness consequent upon hypoglycemia or upon ketosis may contribute to automobile and other accidents

The problems resulting from any accident may become greatly magnified in accordance with the particular state of

the patient at the time Thus trauma with resulting concussion of the brain in diabetic patients who, at the time are already hypoglycemic may have more serious consequences in terms of failure to recognize the hypoglycemia in the first place and secondly in terms of serious damage to the brain with consequent difficulty in treatment and prolonged recovery If the diabetes is unknown and unrecognized and the patient is in an early stage of acidosis defects in vision may lead to serious accidents A study of accidents occurring in diabetic patients at the New England Deaconess Hospital has shown clearly that those patients with diabetes who have received careful instruction in hospital teaching classes have actually had fewer automobile accidents than a control series of non diabetics of similar age and education In the presence of long standing diabetes bony fractures carry special implications Failure of callus formation and bony union often associated with osteoporosis or with difficulty in controlling infection may lead to disaster Without attempting to mention the almost infinite variety of emergency situations which may be affected by the state of diabetes in a given patient two emergencies which by their very nature are diabetic will be here considered namely diabetic coma and hypoglycemia

HYPOGLYCEMIA

In the treatment of diabetes prevention of severe episodes of hypoglycemia is an important problem It is true that mild attacks of hypoglycemia are of common occurrence both in the hospital during treatment and outside the hospital in ordinary practice Indeed it is probably true that a young patient with moderately severe diabetes cannot be kept

under adequate control without occasional periods of hypoglycemia When promptly recognized and treated no harm results On the other hand severe hypoglycemia may have serious consequences affecting judgment success in passing examinations occasionally causing serious accidents The fear of an insulin reaction may be exaggerated so that excessive amounts of carbohydrate are taken and as a result of the positive tests the insulin dose is steadily increased Then too the hypoglycemia may result in excessive glycogen breakdown hyperglycemia again requiring additional insulin It is of the utmost importance that diabetic patients avoid frequent or serious insulin reactions Fortunately almost all insulin reactions are avoidable and preventable provided close enough attention is paid to urine tests and to diet

Fatalities are rare but probably have occurred in much larger numbers than are reported in the literature The fact that an apparently mild reaction may progress rapidly in some diabetic patients to a severe and critical condition makes it necessary to regard every episode of hypoglycemia as in a sense an emergency requiring immediate use of carbohydrate to bring it to an end

SYMPTOMS OF HYPOGLYCEMIA

When an individual develops symptoms of sweating nervousness tremor faintness hunger headache rapid heart action and especially double vision or unsteady gait within a few hours after the taking of insulin he probably is suffering from hypoglycemia In some diabetic patients who have relied upon certain symptoms to warn them of the onset over a number of years there comes a time when either these symptoms are unnoticed or are absent and then they may

become mentally confused or even unconscious without an opportunity of receiving treatment Convulsions are a common symptom in this severe type of hypoglycemia

Patients who receive unmodified or clear insulin may experience a maximum fall in blood sugar and hypoglycemic symptoms 3 4 hours after receiving the dose However such a reaction could occur shortly after taking the dose if the interval between receiving the insulin and the taking of food was prolonged After insulin administration of long duration such as Globin or NPH reactions may occur at longer intervals Thus with Globin insulin about 8 10 hours is the usual interval with NPH insulin 8 10 hours and 12 24 hours after protamine zinc insulin Unusual exercise or an especially meager diet and particularly the omission of a meal or the occurrence of diarrhea, will make more likely the occurrence of an insulin reaction

The symptoms of a reaction are said at times to occur with a high blood sugar or a blood sugar still above normal This must be extremely rare However it must be admitted that if the blood sugar falls very rapidly sometimes symptoms do develop by the time the blood sugar has reached normal or very little below normal Confusion often occurs because patients even after many years of diabetes may think they are having insulin reactions while the exact opposite is true Therefore we make it a rule to take a blood sugar in every patient who thinks that he may be having an insulin reaction In this way we rule out false reactions and are able to help patients in understanding their symptoms when at home

A common cause of confusion arises when the urine shows a strongly positive test for sugar at a time symptoms are present due to the fact that the urine has been in the bladder

several hours during which time the blood sugar has fallen from a high to a low value Patients therefore are warned to test not only the first specimen but to wait a few minutes and test a second specimen of urine which will then be more likely to reveal the fact that blood sugar has fallen provided the bladder was really emptied at the first voiding In cases of doubt a therapeutic test of the reaction may be recovery following the administration of 5 25 grams of carbohydrate Extraordinary variations in the sensitiveness of the central nervous system in hypoglycemia occur The severity of the reaction oftentimes is not closely related to the extent of the hypoglycemia Babies and small children frequently have blood sugar levels of 40 mg or less without recognizable symptoms During severe insulin reactions there may actually be no true glucose in the blood and any blood sugar value of 15 30 mg per cent obtained by methods in common use should be regarded as indicating only *non glucose reducing substances*

The signs and symptoms of hypoglycemia relating to the nervous system may be classified in more than one way Studies of the effects of insulin hypoglycemia used in the treatment of schizophrenia suggest that symptoms may be explained on the basis of the metabolic rate of each region of the brain As the hypoglycemia proceeds symptoms become more severe Five stages described by Himwich concluded with the most dangerous stage characterized by profound unconsciousness respirations are shallow the heart rate slow the pupils contracted and no longer react to light and perspiration with subnormal temperature are present

In patients found unconscious the striking features are that the skin is moist the pulse rate may be rapid and the

blood pressure slightly elevated or normal Twitching or convulsive movements may be present Opisthotonus may occur

TREATMENT OF HYPOGLYCEMIA

In an unconscious patient the immediate administration of a glucose solution preferably in 50 per cent concentration is indicated It is our rule to continue the administration in such patients who are known to have insulin hypoglycemia continuously until the patient arouses Sometimes 50-75 grams of glucose have been necessary To omit glucose administration after the first injection hoping that recovery will take place may be a fatal mistake In some diabetic patients irreversible damage to the brain occurs with extraordinary rapidity Patients may remain unresponsive for days or even two weeks and still recover

In mild reactions the administration of 5 or 10 grams of glucose orally as fruit juice or candy may be effective If the patient will not swallow glucose can be given by intranasal stomach tube or by rectum Glucose corn syrup molasses and honey may all be used Substitutes for glucose such as levulose and cane sugar are not quite so effective

Adrenalin will mobilize glucose in the blood provided the body contains glycogen Consequently the mothers of diabetic children are frequently advised to keep in the house ampules of adrenalin chloride 1 1000 strength and to give by injection 0 3 to 0 5 cc subcutaneously until the child is willing or able to take food by mouth

In the following table is summarized the common causes of hypoglycemia symptoms and treatment Under "Insulin Administration brief mention should be made that occasion

ally the surrepetitious administration of insulin by either a diabetic or non diabetic patient may give rise to hypoglycemia which may be difficult to recognize without a known history Such individuals are usually from the group of nurses physicians or patients familar with insulin and its effects Suicide has been attempted in some cases Islet cell adenomata and carcinomata have been extremely rare in diabetic patients but some 400 non diabetic patients are recorded in whom typical hypoglycemia has occurred due to functioning islet cell tumors

Functional hyperinsulinism is perhaps a common cause of low blood sugar values of 40 to 60 mg 2 or 3 hours after a meal occurring in non diabetic individuals

It should be emphasized that the prevention of insulin reactions in diabetic patients may be accomplished by the adjustment of insulin and diet particularly the use of between meal lunches bedtime treatment and a properly planned diet

TABLE 1 HYPOGLYCEMIA

CAUSES

- I True hyperinsulinism (insulin excess)
  - A Insulin Administration
  - B Hyperactivity of the Islands of Langerhans
  - C Functioning island cell adenomata or carcinomata
- II Relative hyperinsulinism
  - A Lack of precursor substances for blood glucose
    - 1 Diminished absorption from the gastrointestinal tract (undernutrition and prolonged exertion)

2 Disturbances in glycogen storage or mobilization as in Von Gierke's, cirrhosis or other disorders of the liver

B Breakdown or lack of development of regulatory mechanism

1 *Normal newborn or premature infants (transient)*

2 Endocrine imbalance (adrenal and pituitary hypo function or possible relative deficiency of the hyperglycemic glycogenolytic factor of the alpha cells)

3 Intracranial injury especially hypothalamic

4 Idiopathic and other forms including dumping syndrome and "functioning hyperinsulinism

SYMPTOMS

Rapidly falling blood sugar commonly produces symptoms of sympathetic nervous system (adrenalin like) slowly produced decline in blood sugar levels may give few or no warning signs until central nervous system and psychic manifestations occur

a Sympathetic System

1 Hunger and faintness
2 Muscular weakness and tremor
3 Sweating
4 Pulse and BP changes
5 Leukocytosis
6 Vomiting and abdominal pain
7 Numbness of the lips

b Central Nervous System

1 Speech disturbance dilated pupils
2 Jacksonian twitchings
3 Double vision
4 Positive Babinski
5 Aphasia

6 Amnesia
7 Loss of reflexes
8 Tonic or clonic cramps (epileptiform seizures)

c Psychic

1 Drowsiness stupor, may understand but cannot speak
2 Psychoses
3 Hysteria
4 Negativism confusion
5 Depression or anxiety
6 Violence
7 Errors in judgment

LABORATORY

Hypoglycemia with blood sugar below 50 mg following over night fasting or unusual exercise suggests island cell tumor Six hour glucose tolerance curve as diagnostic aid

TREATMENT

For Emergencies (a) conscious patients—sugar, syrup fruit for immediate effect and protein food for continuing reactions (b) unconscious patients—intravenous administration continuously of glucose solution until safe blood sugar level is maintained Fifty per cent solution initially may be followed by five per cent or ten per cent glucose solution Use of adrenalin especially in children If available glucagon 0 5 1 0 cc may be given

Surgical (a) excision of island adenomata or (b) partial pancreatectomy for hyperplasia of Islands of Langerhans

PREVENTION

For diabetics adjustment of insulin and diet. Between meal lunches and bed time feeding

For relative hyperinsulinism diet low in carbohydrate and high in protein and fat

For island cell carcinoma cortisone or alloxan may be given a trial

DIABETIC ACIDOSIS AND COMA

Diabetic coma is the most characteristic feature of diabetes resulting from insulin deficiency and ketosis The word *coma* suggests profound unconsciousness but the patient who is developing diabetic ketosis may be in danger long before he is unconscious Therefore the conventional use of the words *diabetic coma* for early as well as late stages of the condition is well justified Wide variations in severity of diabetic acidosis occur and various standards of definition have been employed For the sake of comparing results in one period with those in another it has long been customary at the New England Deaconess Hospital to classify any case of diabetic acidosis or ketosis as one of diabetic coma when the carbon dioxide combining power (or more recently the carbon dioxide content) of the blood plasma is 20 vol per cent (9 0 milliequivalents per liter) or less Historically this dividing line was accepted shortly *after the introduction of insulin on the clinical* experience that prior to the use of insulin very few patients with diabetic ketosis recovered if the CO content of the blood *was below this accepted figure Deaths from diabetic coma* have become relatively infrequent but they still do occur because of ignorance delay in diagnosis and other factors *Factually* the percentage of admissions in diabetic coma at the New England Deaconess Hospital has steadily de

clined as the total number of diabetic admissions has in creased In recent years, emphasis has been placed upon dis turbances of electrolyte metabolism and especially that of potassium Such changes are of great importance and as re search has improved our understanding appropriate preven tive treatment is now available

Incidence Formerly diabetic coma was the chief cause of death among diabetic patients Before the use of insulin a diabetic child rarely lived for more than two years Nearly 64 0 per cent of all diabetic patients dying in the Naunyn Era from 1897 1914 died in coma This figure fell during the years from 1914 1922 to 41 0 per cent With the increasing use of insulin from its introduction in 1923 the percentage of deaths from coma steadily dropped deaths of patients at the Joslin Clinic during the year 1956 from diabetic coma fell to zero From January 1923 to January 1 1959 we have seen a total of 1002 cases of diabetic acidosis in which the CO combining power or content of the blood plasma was 9 0 mEq /1 or below These patients were all treated as emergencies in the New England Deaconess Hospital Re ports from other institutions and countries clearly indicate that diabetic acidosis is still an important problem first be cause of its immediate emergency character and second because it represents a maximal metabolic disturbance in the diabetic and is clearly related to later complications

Etiology The common causative factors are (1) insufficient insulin or the omission of insulin (2) infections (3) resist ance to insulin effectiveness (4) anesthesia and shock (5) vomiting and diarrhea from any cause *(6) thyrotoxico is*

(7) pregnancy and the toxemias of pregnancy In our experience it is the background of gross dietary errors and lack of control of diabetes which is the most important single factor underlying the development of diabetic coma when the immediate factors mentioned above are superimposed

Insulin Resistance Particularly if infection or myocardial infarction accompanies diabetic coma large amounts of insulin may be required specific antibodies to insulin have been isolated from the blood in patients with diabetic coma by Field and Stetten Insulin dosage in amounts varying from 2 000 to 20 000 units has been given in exceptional patients A wide variety of infections including pneumonia carbuncles abscesses tuberculosis hepatitis and appendicitis have been factors in inducing diabetic coma

In previously untreated patients or patients with insufficient insulin dosage a rapid rise in blood sugar level and glycosuria takes place The loss of calories due to faulty carbohydrate utilization stimulates the utilization of protein and fat Fatty acids are transported to the liver where the formation of ketone bodies occurs A high total metabolic rate occurs and increased fat protein utilization continues As the metabolites of protein and fat combustion are formed in excess these blood levels rise The serum bicarbonate falls as base combines with acid metabolites Large quantities of base particularly sodium potassium are lost in the urine producing acidosis and dehydration Acetone appears first in the urine following its formation from aceto-acetic acid As the ketosis increases diacetic acid itself and finally beta hydroxybutyric acid appear As acidosis advances adrenal function is impaired Large amounts of acetone bodies may accumulate in

the serum even though no large quantities are found in the urine In early stages the total ketone bodies in the blood plasma may be only 25 to 50 mg per 100 cc but in unconscious patients values usually will exceed 100 mg per 100 cc

Lack of previous diagnosis and treatment of diabetes must still be emphasized in the etiology of coma At the New England Deaconess Hospital from year to year from 12 0 to 16 0 per cent of our cases had no diagnosis of diabetes until the onset of acidosis Likewise in the series of Zieve and Hill Loeb and Dillon and Dyer from 17 0 to 30 0 per cent of the patients were ignorant of their diabetes at the time of acidosis

DEVELOPMENT OF DIABETIC COMA

The onset of diabetic acidosis and coma may be gradual and vague or it may be rapid Symptoms may have been overlooked by the patient and sometimes even the physician may be misled Particularly in newly discovered diabetes in youth almost any sign of illness should be regarded with apprehension and acidosis considered Nausea indigestion headache and particularly fever due to mild infection should be regarded with suspicion

Patients should be given instructions early in the course of diabetes Preventive measures can do no harm and if adopted early may avert acidosis in most cases except those rendered desperate by severe complications Instructions commonly given to patients include the following (1) go to bed (2) keep warm (3) test the urine for sugar and notify your doctor (4) secure a nurse or someone to help you and conserve your energy (5) move the bowels by enema (6) take a glass of liquid each hour such as coffee tea or broth, and

(7) especially important is the rule to continue insulin even if vomiting unless the urine test is sugar free or a physician advises to the contrary

SYMPTOMS OF DIABETIC COMA

There are no pathognomonic symptoms in early diabetic coma but labored respiration with characteristic deep air hunger as described by Kussmaul and drowsiness are outstanding Increased glycosuria with increased thirst and particularly the demonstration of large amounts of acetone in the urine using the tests for acetone which are taught to patients give warning Then headache malaise nausea abdominal pain and particularly severe constipation are frequent Abdominal pain may be a very prominent symptom sometimes accompanied by other symptoms suggesting a surgical complication

The stage of nausea and vomiting which is so common may be followed by drowsiness then stupor and finally coma so severe that almost no painful stimulus will be recognized Fortunately today most patients are recognized and brought to a hospital before this final stage of unconsciousness has been reached The patient may be in grave danger long before drowsiness or unconsciousness has occurred Severe acidosis may be present with a reduction of the CO combining power to values well below 8 mm and yet the patient especially if he is young may walk into the hospital

The patient in fully developed diabetic coma presents a group of findings which often is characteristic He lies semiconscious or unconscious and completely relaxed although in some cases there may be a good deal of activity The skin is dry notably the axillae The face is flushed and drawn the

feet and hands cold Respiration may be slow or rapid but its depth is frequently striking Nevertheless it must not be forgotten that a patient may have passed through this early stage of Kussmaul respiration and then in exhaustion respirations may be shallow The eyeballs are soft to the touch and care must be taken not to press quickly for fear of injury to the retina The mouth and tongue are usually extremely dry vomiting occurs frequently Abdominal findings may include evidence of a distended stomach but this condition may easily be missed The muscle spasm and pain may suggest acute appendicitis The pulse is rapid and weak and the blood pressure low In far advanced cases the pulse may be imperceptible and the blood pressure unobtainable Usually the muscles are flaccid and the tendon reflexes absent The rectal temperature may be subnormal If active infection is present as treatment proceeds the fever of a hidden pneumonia pyelonephritis or phlebitis may return

LABORATORY FINDINGS IN DIABETIC ACIDOSIS

Blood Sugar High blood sugar levels are characteristic of diabetic coma In 945 cases we have seen 40 patients in whom the initial blood sugar was 1 000 mg per 100 cc or above The highest level with recovery had a value of 2 250 mg per 100 cc However many patients who enter the hospital in severe acidosis not yet in coma may have blood sugar values of 350 to 500 mg per 100 cc the average blood sugar on admission to the New England Deaconess Hospital for example has varied in series to series between 499 and 581 mg per 100 cc The level of the blood sugar is of considerable significance since it is a measure not of the food eaten by the patient but of the degree of intracellular disturbance

and excessive glucose formation from protein and fat as well as from stored glycogen Percentages above 4 or 5 are uncommon and of good omen When the glycosuria is reduced to less than 1 0 per cent it frequently means failing renal function The showers of granular casts seen in the sediment are a classical finding in diabetic coma even in children

Blood electrolytes These are seriously disturbed during diabetic ketosis and this disturbance in electrolyte metabolism may itself be the cause of death in patients whose insulin treatment has been skillful Electrolyte depletion particularly of sodium and most important of potassium occurs during the course of diabetic acidosis and requires special handling Salt depletion has been known as a feature of diabetic acidosis for many years

THE DIFFERENTIAL DIAGNOSIS OF COMA

An unconscious patient always presents a serious diagnostic problem If the patient is a known diabetic the possibility of acidosis and coma may be the first thought but it must also be followed by a second consideration of various other possible causes *In small children coma may be the first evidence of diabetes* In the following table are summarized the *chief points in distinguishing between hypoglycemia and* diabetic coma Acetonuria develops rapidly in diabetic patients and may occur in non diabetic patients from simple starvation

1 *Hypoglycemia* One of the most serious errors in internal medicine is the administration of insulin to a patient who already has hypoglycemia If a patient is known to have diabetes

DIFFERENTIAL DIAGNOSIS IN HYPOGLYCEMIA AND DIABETIC COMA

The features of an insulin reaction listed in this table are those observed after rapidly acting insulin regular or crystalline In certain respects reactions due to the slowly acting protamine zinc insulin may differ Headache particularly occipital nausea and even vomiting may occur These symptoms make the differential diagnosis between reactions from protamine zinc insulin and coma more difficult than that between regular or crystalline insulin and coma Reactions following NPH Lente or globin insulin resemble more nearly those of regular insulin

	Hypoglycemia	*Diabetic Coma*
History	Insufficient food excess insulin excess exercise	Insufficient insulin infection gastrointestinal upset
Onset	Following short acting insulin Suddenly a few hours after injection Following long acting insulin Relatively slower many hours after injection	Slow hours or days
Course	Anxiety sweating hunger headache diplopia incoordination twitching convulsions coma (Headache nausea and haziness especially following long acting insulin)	Polyuria polydipsia anorexia nausea vomiting labored deep breathing weakness drowsiness possibly fever and abdominal pain coma
Physical findings	Pale moist skin full rapid pulse dilated pupils normal breathing blood pressure normal or elevated overactive reflexes positive Babinski	Florid dry skin Kussmaul breathing with acetone odor decreased blood pressure weak rapid pulse soft eyeballs
Laboratory findings	Second urine specimen sugar and ketone free low blood sugar normal serum CO_2	Urine contains sugar and ketone bodies high blood sugar low serum CO

and known to be taking insulin hypoglycemia should always be considered if the patient is found unconscious semi conscious and particularly if he has been convulsive Patients in hypoglycemia are usually sweating or the skin is moist but there are exceptions Preceding mental behavior may have been that of simple confusion obstinance or other manifestations of cerebral reaction A urine specimen obtained by catheter should be sugar free or else nearly so unless the urine shows sugar because it has been long in the bladder Hypoglycemia from protamine zinc insulin is frequently more gradual than with regular insulin and may require more prolonged treatment If a patient is found unconscious at home or at a long distance from a laboratory a small amount of glucose in sterile solution may be given intravenously If complete recovery is prompt then an insulin reaction is probable However such a response should be definite if the assumption that the condition is due to hypoglycemia is correct

2 *Uremia* The problem of the differential diagnosis of uremia is today more frequent than ever since more diabetic patients are living longer and are reaching the stage where because of uncontrolled diabetes of years duration diabetic nephropathy is present If albuminuria is marked nitrogen retention and uremic symptoms may occur The blood sugar level may be extremely high and the CO_2 content of the blood depressed as in diabetic ketosis the blood level for acetone will be normal It is the patient however, with both diabetic ketosis, as shown by these findings and also renal failure who will require careful laboratory study and great care in treatment

3 *Cerebrovascular Accidents* These may have a relatively sudden onset or may have been preceded by severe headache and some hours of symptoms The spinal fluid may contain gross blood Little or no acetonuria may be present

4 *Toxicity* From such causes as acute infection drug poisoning brain tumors or meningitis toxicity must be considered

THE TREATMENT OF DIABETIC COMA

1 *Treatment at Home* Diabetic acidosis is so serious a problem both in its treatment and in the diagnosis of accompanying complications that the patient should be hospitalized Here every laboratory facility and uninterrupted treatment should be available including the administration of fluid and frequent blood analyses It is customary to give insulin immediately at home provided the diagnosis seems certain Doses ranging from 50 200 units have been given prior to admission and no serious errors have been made The decision however is an important one and the size of the dose may be a matter of some concern Prior to arrival at the hospital preparation should be made including equipment for administration of solutions intravenously A portable diabetic treatment cart equipped with such materials has been found most useful at the Deaconess Hospital Laboratory technicians services should be available immediately and continuously both day and night in the emergency department In Table II is summarized the plan of treatment which has been found useful at the New England Deaconess Hospital

Our objective has been to give the optimal amount of insulin required in the given case as quickly as possible The problem is to determine what is the needed dose in the individual patient A child or young adult with recently developed diabetes may require relatively small doses of insulin for an immediate effect On the other hand a patient whose diabetes is of long duration and who has taken large doses of insulin for several years may not have become resistant and require a dose of 500 or 1 000 units If delay occurs in assessing the severity of the patient the opportunity for recovery may have been lost

On admission as soon as the diagnosis has been made physical examination performed and urine tested the preliminary dose of insulin is given At present in severe patients we give at least 50 per cent of the initial dose intravenously and in the most severe types give all insulin intravenously during the first few hours This insulin should be of the unmodified or regular type Protamine zinc insulin acts so slowly that it should not be used except as an adjunct to treatment In Table III is summarized the relationship between the blood sugar levels on admission and the insulin requirement It is apparent in recent years that approximately 50 per cent of the insulin required in the first 24 hours should have been given during the first 3 hours after admission if treatment has been sufficiently aggressive and clinical acumen reasonably satisfactory It is worth pointing out however that in the future the diabetic patient who has diabetic nephropathy may have a very high blood sugar but will not be really insulin resistant Uremia is often accompanied by sensitiveness to insulin and therefore the dosage of insu

lin under such conditions must be smaller in amount and perhaps given somewhat more frequently

2 *Fluids* Dehydration and loss of electrolytes two of the most important clinical features of diabetic coma and the subjects of exhaustive study in recent years, are clearly depicted in the appearance and condition of the patient The dry inelastic skin the dry brown tongue and soft eyeballs together with falling blood pressure are characteristic features Restoration of fluid and electrolytes is an objective only less important than the proper administration of insulin

TABLE II TREATMENT OF DIABETIC ACIDOSIS AND COMA

Diabetic acidosis is the result of insulin deficiency Nausea *vomiting abdominal pain dehydration shock air hunger drowsiness* lead to coma and (if insufficiently treated) death Important chemical features are hyperglycemia glycosuria, ketonemia ketonuria reduction of plasma CO depletion of electrolytes especially potassium

FIRST HOUR AFTER ADMISSION

Special nurse preferably experienced in coma treatment for the first few hours

LABORATORY

1 *Urine* Examine for sugar acetone diacetic acid albumin coma casts and pyuria Catheterize if necessary
2 *Blood* Test for sugar CO content and non protein nitrogen with emergency report within an hour White

blood count Serum potassium and amylase levels Hematocrit as aid in assessing dehydration

CLINICAL

3 *Search for complications and establish diagnosis*
  - A History to explain cause of coma
  - B Physical examination noting particularly—
    - (a) State of consciousness type of respiration pulse rate blood pressure and rectal temperature
    - (b) Look for soft eyeballs dry tongue dilated stomach cold and mottled skin impacted rectum and tendon reflexes
  - C X ray chest and abdomen when possible
  - D ECG (a) coronary (b) potassium changes

4 *Insulin* 50 to 100 units of regular insulin (one half dose intravenously) at once for adults In severe cases especially with circulatory collapse give all insulin intravenously If blood sugar exceeds 300 mg per 100 cc and if the blood CO_2 content is 9 millimols per liter (20 volumes per cent) or less the dose will need to be repeated The insulin dose would be proportionately less (20 to 40 units) in young children especially if diabetes is of recent onset In cases with blood sugar between 600 and 1000 mg give 200 units additional and with blood sugar over 1 000 mg give 300 units additional

5 *Gastric Lavage* Aspirate completely and wash stomach with warm water with greatest care

6 Normal saline intravenously 2000 cc It is desirable to change to a solution of saline lactate after the first liter of saline solution is given (to 700 cc saline add 1 ampoule (40 cc) 1 molar lactate and make up to 1000 cc with sterile distilled water) If lactate is unavailable nor

mal salt solution may be continued Avoid too rapid administration especially in older patients

7 Keep patient warm yet avoid burns as from hot water bottle

SECOND TO SIXTH HOUR

The gravity of the case may require repetition of first hour's total insulin in the second hour

8 Give potassium solutions by vein for definite indications (a) when blood analysis or ECG clearly indicates hypokalemia (b) when potassium depletion is probably present as result of prolonged serious ketosis and/or deficient potassium intake (c) only in the presence of adequate urinary output 25 meq K per hour up to 100 meq may be given

9 Repeat blood sugar and CO determinations after two or three hours For rising blood sugar give insulin hourly 50-200 units or more according to estimate of prognosis and hourly blood sugar tests

10 Fluids by mouth (as soon as tolerated) limited to 100 120 cc per hour of broth ginger ale orange juice tea or coffee hourly to be sipped by patient or spooned by nurse For children limit to 50 cc per hour at first Then if nausea and vomiting recur withhold fluids orally for 2 to 6 hours (lavage stomach again if indicated) and then resume

11 *Enema* for cleansing and to relieve abdominal distension

12 Record and note changes in blood pressure pulse and temperature hourly Consider vasopressor drugs for transfusion if in deep shock

13 Urinalysis for sugar and diacetic acid every hour Re

cord hourly output as index of dehydration and renal function

14 Antibiotics (parenteral) as penicillin streptomycin or tetracyclines frequently needed when blood pressure normal

15 *Urinary output* Record hourly and note with alarm any sign of oliguria 1500 cc intravenous saline lactate for persisting shock Repeat as necessary For anuria associated with hypochloremia give 50 cc of 10 per cent salt solution intravenously Beware producing excessive diuresis with consequent loss of base especially of potassium (by too rapid administration of I V Fluid) For anuria associated with hyperchloremia omit all saline using only glucose in water intravenously Volume of such fluids may need limitation to 100 cc in 24 hours

SIXTH TO TWENTY FOURTH HOUR

16 Repeat blood sugar and CO determinations Give insulin 50 200 units if blood sugar and CO levels are not improving Insulin (regular) may be given according to urine tests every 1 to 4 hours if fall in blood sugar has been satisfactory

If test is—	Red	Orange	Yellow	Green	Blue	
Give —	20	16	12	0	0	units

For young children give half dose

17 Soft or liquid food such as oatmeal gruel orange juice or milk diluted half and half with lime water *not to exceed* 10 gm carbohydrate per hour Glucose (5% in saline) I V at rate of 200 cc per hour only when blood sugar approaches normal

18 Sudden onset of muscular weakness or loss of tendon reflexes and shallow respiration suggest hypokalemia Potassium may be given p o or i v if changes in ECG or in serum potassium are present

SECOND DAY AND SUCCEEDING DAYS

19 Soft food—Diet carbohydrate 100 to 150 gms protein 50 gms fat 50 gms Gradually return to standard diabetic diet for age and weight with carbohydrate 150 to 200 gms protein 60 to 100 gms fat 60 to 120 gms daily

ADDITIONAL NOTES

I Differential diagnosis should include the acidosis of diabetic nephropathy occurring in patients with diabetes of long duration Uremia may result in retention of ketone bodies in the blood plasma although they may be absent or reduced in concentration in the urine Examine plasma for acetone by nitroprusside test* or quantitate ketone bodies in blood

TOTAL KETONES IN BLOOD

	Mg Per 100 cc
Normal	0 to 5
Non Diabetic Uremia	5 to 60
Diabetic Coma	50 to 200+

Plasma Acetone Test
4 cc of blood in an oxalate tube centrifuged until clear plasma obtained Make solutions of 1 in 2 1 in 4 and 1 in 8 with normal saline or tap water Place 3 drops of undiluted plasma and the three dilutions on separate small mounds of acetone test powder At the end of 60 seconds read the color (do not allow to stand longer) Depth of purple color indicates concentration of acetone and in some cases may be used as a clue to insulin resistance

II To avoid pulmonary edema rarely exceed 5 000 cc parenteral fluid in 24 hours and check frequently for signs of edema If urinary output exceeds 40 cc per hour after parenteral fluid has been given up to 3 000 cc grave dehydration no longer exists

III Electrolyte containing solutions Potassium should not be given intravenously in excess of 25 mEq per hour!! Rarely is it wise to exceed 100 mEq in 12 hours unless definite hypopotassemia is present and urine excretion is ample After 12 to 24 hours if 3 to 4 grams potassium cannot be taken by the patient in diabetic diet a simple solution may be taken in divided amounts Thus two hundred cc orange juice plus 2 grams potassium phosphate may be diluted with water to 500 cc *Of this give 100 cc per hour With fall in* blood sugar and need for potassium a 5 cc ampule (2 grams dibasic potassium phosphate and 0 4 grams monobasic potassium phosphate) may be added to 1 000 cc of 5 per cent glucose for intravenous administration if indicated

IV Electrocardiographic signs of

A Low serum potassium (below 3 0 mEq)
  1 Lowered or inverted T waves
  2 Depressed ST segments
  3 Lengthened QT or appearance of U wave
  4 Prolonged P R interval

B High serum potassium (above 6 0 mEq)
  1 High peaked T waves
  2 Wide QRS
  3 Disappearance of P waves
  4 AV dissociation
  5 Final disorganization of ECG

Note A normal ECG does not exclude K deficiency The above changes may not always be due to hypokalemia

As indicated in the outline the restoration of fluid and electrolytes is easily begun by the immediate intravenous administration of physiologic salt solution The amount which may be ultimately required is often difficult to determine since actual measurement of the degree of dehydration is uncertain In the presence of anuria or a declining urine output with or without infection a maximal degree of dehydration may be presumed present In the average patient from 3 000 to 500 cc of fluid is necessary in the first 6 10 hours In some patients as much as 10 000 cc has been necessary providing it is determined that the oliguria is due to dehydration alone During the first few hours of treatment no glucose solution may be given It is clear that in these first few hours of treatment the prime objective is to determine the presence or absence of insulin resistance If the blood sugar level is maintained by glucose administration then the decline in blood sugar may not be used as an index of the insulin requirement If levulose is used it is possible for serious hypoglycemia to be produced by giving too much insulin since the presence of levulose in the blood would give a high blood sugar value although the true glucose value in the blood may be at a seriously low level An important point with respect to glucose is that when large amounts of insulin are being used the more glucose that is given the more potassium will be withdrawn from the blood and deposited with the glycogen Potassium is administered in coma patients when the history indicates that depletion of potassium is present by reason of long continued lack of

diabetic control or acidosis itself or when serum potassium determinations or the electrocardiogram indicates a low serum potassium value

3 *Gastric Lavage* An important measure in preparing the gastrointestinal tract to receive food which is an important objective in the treatment of diabetic coma is the use of gastric lavage It should be carried out routinely unless the patient is in such grave condition that the procedure involves some risk Sometimes one is surprised by the finding of considerable quantities of fluid food remains and old blood The removal of such contents and lavage relieves distress stops vomiting and prepares the way for the taking of food by mouth

4 *Blood Transfusions* Transfusions have been used with few of our patients except those in whom there has been a known loss of blood as in gastric hemorrhage The use of transfusions to combat circulatory collapse may be utilized Usually however the administration of adequate fluid and electrolytes has been sufficient

5 *Glucose and Food* The use of glucose or fructose depends upon the period in the treatment of diabetic coma under consideration The treatment of diabetic acidosis or coma may be divided into two periods (A) The first 3 8 *hours* after admission of the patient when marked hyperglycemia and dehydration are present This period may be very short as little as two hours in children or patients with diabetes of short duration and may be much longer *in severe cases* (B) The time when the effective action of a sufficient dose of

insulin has produced a decline in blood sugar levels roughly of 40-60 per cent from the original level

In the first period (hyperglycemia and dehydration) the administration of glucose cannot do any possible good It has never been shown in a patient in diabetic coma with a hyperglycemia of 500 1 000 mg per cent, that the utilization of glucose is increased by the administration of glucose It is only the effective action of insulin which brings about an increase in intracellular glucose utilization

In the second period when blood sugar is falling the use of glucose has always since the introduction of insulin been a necessary procedure It is then that hypoglycemia actual or impending is being treated It must be emphasized again that when large doses of regular clear insulin are given the duration of insulin action is greatly increased from the usual 6-8 hours to 18 24 hours Therefore when the blood sugar has fallen significantly in a period of 4 5 or 6 hours the addition of further large amounts of insulin must be carefully considered lest severe hypoglycemia be produced

The administration of potassium containing foods should begin as soon as the patient is able to take food by mouth Sometimes this is within 4 8 hours It is important that small amounts of fluid be given by mouth in the patient who has had a dilated stomach Usually we aim at 10 grams of carbohydrate per hour using hot fluids such as thin oatmeal gruel or orange juice diluted half and half with warm water Obviously in unconscious patients intravenous administration of glucose may be necessary during the first few days of treatment The use of fructose or invert sugar has been urged on the concept that fructose may enter the glycolytic cycle without the aid of insulin Fructose forms glycogen

easily However possible gain is limited by the fact that only a small portion of the fructose escapes conversion to glucose

6 *Other Remedies* The use of vitamins and antibiotics is routine in the unconscious patient who may be suffering from a hidden infection and who may because of the severe metabolic disturbance be in danger of developing a diabetic neuropathy Preparations of B Complex are commonly given in parenteral fluids

A cleansing enema should be routine in patients with any severe acidosis unless diarrhea or some other complication is present

Alkalies have not been given in the form of soda bicarbonate or other similar preparations for years at the Deaconess Hospital It is seldom that one hears emphasis placed upon their value in comparison with the use of insulin and other measures

7 *Convalescence* Recovery from acute acidosis may be followed by a return to normal which is steady but may be prolonged It is important to remember that patients long unconscious or who have been in severe shock may not be able to take much food or to return to a full diet for a week or two In some patients severe neuropathy follows diabetic coma and this neuropathy occasionally is fatal in other cases many months are required for recovery

The Level of Blood Ketones and the Blood CO_2 These are markedly affected as acidosis progresses In early stages acetone may be found both in the serum and in the blood in considerable amounts *before the CO of the blood*

changes In advanced acidosis however the CO content of the venous blood declines and usually is below 8 o mEq in unconscious patients Although a general relationship between the state of consciousness and the depletion of CO of the blood and the level of ketones exists there are however notable exceptions

The Nonprotein Nitrogen of the Blood This is frequently elevated to 50 or 75 mg during acidosis partly due to the increased protein breakdown of diabetic acidosis and partly to beginning renal failure Levels above 100 are frequently associated with some severe renal lesion

The Urine Almost always the urine shows large quantities of diacetic acid by the ferric chloride test and also acetone As coma progresses and renal failure develops with or without shock the excretion of acetone bodies is greatly reduced and the urine may become negative However in these cases the blood plasma will invariably show a positive test for acetone The quantities of acetone in the blood plasma using acetone test powder are described under the Table outlining treatment The amount of sugar in the urine varies considerably

COMPLICATIONS OF DIABETIC COMA

Complications independent of diabetic ketosis may seriously affect the rate of development of coma its course and may actually determine the outcome Infections are the most frequent complication but others such as myocardial infarction or a ruptured disc have occurred

Hypoglycemia may occur especially if an error in judg

ment results in excessive dosage with insulin The patient may pass from the condition of diabetic coma to one of insulin hypoglycemia without very striking physical signs Therefore frequent blood sugar estimations are invaluable and indeed their availability constitutes one of the important reasons for the treatment of such patients in a hospital However with certain precautions it has been the experience at the New England Deaconess Hospital that hypoglycemia during and after the treatment of diabetic coma has been almost unknown This is largely due to our deliberate plan of preparing to feed the patient small amounts of carbohydrate every hour as early as the gastrointestinal tract will permit such treatment

The recurrence of diabetic coma after apparent recovery is not unknown but should never happen in a well regulated hospital

Circulatory collapse with low blood pressure rapid pulse and oliguria may occur as a late event at any age but is much more frequent in older patients Recovery may occur in a pulseless patient *If the collapse is merely the terminal stage of dehydration then the use of sufficient fluid will be effective* Transfusions of whole blood in some patients may be useful The supplementary use of Adrenalin or Ephedrine and particularly Levophed has been most helpful

Anuria or oliguria is an ominous complication in ketosis It is essential to chart the urine output of the patient in diabetic coma hour by hour and to view with alarm any decline in the rate of urine secretion Under these circumstances determinations of serum electrolytes particularly with reference to potassium and sodium may give the clue for therapy Anuria persisting for days may still terminate in recovery

The following causes have been observed in one or more of our coma cases in whom anuria was present (a) dehydration and circulatory collapse the most frequent (b) pyelonephritis acute or chronic (c) acute renal injury as in necrotizing papillitis or the hepato renal syndrome (d) acute glomerulonephritis often indicated by history of facial edema and smoky urine (e) shock from burns or trauma or myocardial infarction, (f) congenital absence of kidney (g) obstruction from stones sulfonamide crystals or tumor (h) acute renal shutdown a rare event occurring even in early ketosis

In contrast to the large amounts of fluid required where dehydration is the chief cause of anuria the treatment in acute renal shutdown is aimed to avoid pulmonary edema and to protect renal tissues by giving intravenous glucose solution in limited quantities Thus, glucose solutions of 20 to 40 per cent concentration in water in amounts varying from 750 to 1200 cc per 24 hours have been employed

THE PROGNOSIS IN DIABETIC COMA

In general the prognosis for the patient with diabetic ketosis depends upon (1) the severity of the acidosis (2) the duration and degree of unconsciousness (3) the age of the patient (4) cardiovascular renal status of the patient (5) complicating conditions (6) grossly abnormal laboratory findings It is important to remember that complications or associated infections may cause the patient s condition to be extremely grave long before he is unconscious Indeed in some series of patients the degree of unconsciousness has been of much less importance in prognosis than these other conditions

Patients in whom acidosis is far advanced have a much more serious prognosis than an individual who is in early acidosis thus the level of CO in the blood like the pH is an important factor In our series of cases the unconscious patient formerly had a much worse prognosis than has been true in the last 10 years Undoubtedly unconscious patients in the last few years have been treated with much greater vigor and much larger doses of insulin used during the first 2 or 3 hours of treatment a factor of prime importance although the availability of the antibiotics for the management of latent or unrecognized infection together with other factors has contributed to reducing the mortality of unconscious patients from 35 per cent to the present 10 per cent The age of the patient is extremely important Nearly 40 per cent of our coma cases occur in patients whose diabetes began in the first 20 years of life although they may be many years older when coma is observed They have a greater advantage in comparison with older patients in whom cardiovascular complications are present Indeed the cardiovascular status in the middle aged and older patients is frequently a determining factor The presence of diabetic nephropathy (Kimmelstiel Wilson) increases the danger

Laboratory data of importance include not only blood sugar levels and non protein nitrogen of the blood but measurements of the degree of ketosis The bedside method described under our rules in the treatment of coma is useful When possible, the actual quantitative determination of the ketone bodies in the blood is of value However the time required for this determination makes it of doubtful value in most hospitals

The causes of death in diabetic coma are varied However

uncomplicated coma is still a cause of probably one third of the deaths due to delay in diagnosis and often to the unfortunate omission of insulin at a time when acidosis was beginning and causing inability to eat Next comes sepsis and metastatic infection Pancreatitis probably occurs more frequently than is appreciated As we make use of the determination of amylase in the blood the frequency of acute pancreatitis during diabetic coma becomes more impressive As a cause of death proven at autopsy it has not been frequent Miscellaneous causes are first cardiac with coronary disease in the background of the deaths which number about one third of all fatalities Other causes are cerebral hemorrhage pulmonary embolism and nephropathy

TABLE III DIABETIC COMA (329 *CASES*)
1946 1958

Admission Blood Sugar Mg /100 cc	Cases No	Average Insulin First 3 Hours Units	Average Insulin First 24 Hours Units
--50	1	325	1125
1300 1600	7	552	1148
1000 1300	20	453	767
600 1000	67	322	499
400 600	115	189	_68
200 400	94	111	154
100 200	5	62	1_8

Low values due to insulin given en route

Part V

THE ACUTE RENAL SYNDROMES

A\ A R Spiegel man M D

XXXI | THE ACUTE RENAL SYNDROMES

In the present state of statistical classification of the causes of death mortality data underestimate the prevalence of neph ritis in the population Because of changes in the procedures of reporting and classifying detailed causes of death many of the death rates of recent years are not comparable to those of earlier years These procedural changes reduce appre ciably the recorded deaths from chronic nephritis However it has been estimated that each year perhaps as many as one per cent of the population in their late sixties become neph ritic Uremia or hypertension with its complications may be the consequences

UREMIA

Uremia is a symptom complex associated with an abnor mally high urea nitrogen and reduced urea clearance Any congenital or acquired lesion which causes destruction of the renal parenchyma to the extent where the remaining tissue

Urine The urine of renal failure is not characteristic it varies with the underlying disorder The patient with glomerulo nephritis may have very little proteinuria, few red blood cells or white blood cells in the sediment At times it is difficult to distinguish this urine from that of pyelonephritis In the nephrotic syndrome on the other hand the proteinuria is heavy and the sediment is loaded with casts and double refractile bodies

Blood The blood findings in early renal failure include an elevation of urea nitrogen creatinine nonprotein nitrogen and uric acid The urea/NPN ratio increases as renal failure progresses Creatinine is a more accurate indication of the intensity of renal failure than is urea because it is not influenced by variations in the protein intake When renal failure is severe the blood creatinine may rise from the normal of less than 2 mg per cent to 10 mg per cent Muscle wasting may account for the rise to some extent The total serum protein is decreased and cholesterol increased especially in the nephrotic syndrome Phosphate is increased and calcium decreased Serum chloride potassium sodium and CO combining power are normal Progressive renal damage results in the accumulation of acid metabolites in *the blood and* the *CO combining power* is then lowered The acidosis may be increased by *sodium* loss consequent upon the failure of tubular reabsorption Usually this does not occur unless the patient voluntarily restricts his sodium intake Later glomerular filtration is impaired and sodium is retained The plasma sodium level would be expected to rise but this does not always follow Plasma sodium may actually be lowered

because the metabolic disturbances may allow sodium to diffuse across the cell membrane into the intracellular compartments By a similar mechanism *potassium* may be increased or decreased in the plasma In acute renal failure however hyperkalemia is a frequent finding and often a cause of death

Phosphorus This is increased because of poor excretion and by a mechanism not well understood serum *calcium* is reciprocally decreased The reduced calcium stimulates the parathyroids and the resultant hyperfunction causes the release of calcium and phosphate from the bone

The electrolyte pattern in renal failure is shown in the table below

Renal F ilur	Sod um	Potas um	CO	Chl ride	Pho ph t	Cal um
Acute	D c sed	Usually incr ed occas ally Norm l	D cr sed	D creased	D cre ed	Usually un h nged
Ch om	N rmal d eased	Normal o d cr sed	D cre ed	Normal o decr as d	I cr sed	Decr ased

DIAGNOSIS OF UREMIA

The diagnosis of uremia is difficult without laboratory assistance The coma of diabetes mellitus cerebral thrombosis and hemorrhage may mimic uremia A normal urea nitrogen signifies that uremia is not the cause of the symptoms A high urea nitrogen is less significant it may occur with cardiac decompensation or cerebro vascular accidents in patients with essential hypertension The diagnosis is even more confusing when uremia and hypertensive encephalopathy

co exist Epileptiform convulsions and loss of vision in a patient whose cerebrospinal fluid pressure is high suggest that the symptoms are on a hypertensive rather than on a uremic basis

Specific gravity of the urine may have differential significance The uremic patient has an oliguric urine of low specific gravity specific gravity of 1020 or more in the presence of oliguria makes the diagnosis of uremia unlikely Exceptions are acute glomerulonephritis and prerenal azotemia where impaired glomerular filtration with normally functioning tubules produce an oliguric urine of high specific gravity

EXTRARENAL UREMIA

In renal uremia azotemia is due to reduced urea clearance resulting from decrease in the size of the total glomerular filtering surface In the extrarenal uremias there is no demonstrable renal lesion urea retention is caused by a decrease in the rate of renal flow Oliguria is prominent in both Differentiation depends upon the specific gravity of the urine In the absence of clinical evidence of acute glomerular nephritis or acute tubular nephrosis a normal specific gravity indicates uremia of extrarenal origin

Extrarenal uremias may be *prerenal* or *postrenal* The prerenal uremias may be due to hypochloremia and hyponatremia resulting from vomiting and diarrhea dehydration which also may be due to vomiting and diarrhea or sudden fall in blood pressure as in circulatory peripheral collapse or congestive heart failure The postrenal uremias are due to obstruction in the urinary outflow tract

TREATMENT OF ACUTE RENAL FAILURE

During the first few days of acute renal failure when oliguria and anuria are the predominant clinical symptoms, the chief hazard is the accumulation in the blood of protein catabolites the most important of which is potassium The principal objective is to diminish this catabolism This may best be accomplished by withholding protein and providing a diet entirely of glucose and fat both of which are oxidized to water and CO_2 Glucose in these patients will have three effects (1) it has a direct protein sparing effect (2) it diminishes the tendency to ketosis and (3) it decreases the liberation of intracellular potassium The maximum protein sparing effect of glucose is provided by 100 gm /day This amount halves the protein utilization and prevents ketosis Increasing glucose administration above this level provides a greater caloric intake but spares little more protein Usually these patients are under some form of stress and require more than the calories provided by 100 gms of glucose To further increase the caloric intake fat is added to the diet Infusions of fat and glucose have been used but pyrogenic reactions have made them impractical

Nausea and vomiting frequently prevents oral feeding Thorazine may be successful in controlling these symptoms should this fail intravenous feeding becomes necessary Administration of an adequate number of calories becomes a problem because of the limited fluid allowance To handle this complication 50 per cent glucose in water may be given intravenously a plastic catheter should be inserted into the brachial or cephalic vein of the arm or the saphenous vein of the leg and threaded into the vena cava Fifty per cent

glucose solution to which 50 mg of heparin has been added is given by the constant drip method The rate of infusion should not exceed 0 4 gm/kilo/hour for optimal utilization This provides the patient with 200 gms of glucose in 400 cc of water daily more fluid may be added if indicated Treatment is continued until diuresis occurs The catheter may be left in place for 5 to 7 days Merrill reports no major embolic phenomena with this technique provided adequate heparinization is maintained

Water Intake It may be difficult to determine how much fluid is to be given to an oliguric or anuric patient As a rule fluid intake should be slightly less than the fluid lost in order to prevent pulmonary edema Daily fluid allotment should be calculated from the extrarenal water loss plus the urinary volume of the previous day to which is added any fluid lost as vomitus diarrhea sensible perspiration and fever In adults the extrarenal water loss is about 1100 cc daily (600 cc through the skin 400 cc through the expired air and 100 cc in the feces) On this regimen the patient should lose weight, if there is no weight loss the fluid allowance should be reduced This calculation does not include water formed in the processes of metabolism and therefore may exceed the requirements Daily weights provide the most accurate measure of effective hydration

Sodium The hyponatremia of acute renal failure may be due to a previous low intake the loss of sodium dilution by excessive administration of water or a shift from the extra to the intracellular spaces In the first two instances the administration of sodium will correct the deficit in the last two re

striction of water is indicated Where several factors are concerned in the production of hyponatremia the administration of sodium may be indicated only if a negative balance can be demonstrated If a negative balance is questionable sodium may be given carefully weighing the benefits to be derived from the correction of the hyponatremia against the possibility of causing pulmonary edema If serum sodium determinations are available the amount of sodium chloride necessary to correct the hyponatremia may be roughly estimated by multiplying the extracellular fluid volume by the deficit in mEq/liter The extracellular fluid volume and plasma volume are difficult to measure In a man of average size the extracellular fluid volume is about 16 liters the plasma volume is about 4 liters These figures will vary with the size of the individual The infusion should consist of 200 cc of 3 or 5% sodium chloride If the loss of sodium is greater than that of chloride or if excessive acidosis exists sodium lactate or bicarbonate should be used instead of chloride When using concentrated solutions about 1/3 of the estimated volume of fluid is given over a period of 3 hours The patient is observed for about 6 hours and if there are no cardiovascular complications a second third is given slowly Improvement will ordinarily occur before the serum sodium returns to normal If there is no improvement after the second infusion the administration of sodium should be stopped

Sodium chloride is not the preparation of choice if the *loss of chloride exceeds that of sodium* This may occur after severe and continued vomiting Hypochloremia is unimportant since its only role in acute renal failure is in the maintenance of acid base balance It therefore requires no

treatment unless retention of sodium results in hypochloremia alkalosis This may be corrected by the administration of 200 cc of 2% solution of ammonium chloride intravenously The ammonium radical is converted to urea in the liver and may cause a slight but insignificant rise in urea nitrogen

Potassium The oliguria or anuria of acute renal insufficiency may result in acute hyperpotassemia Potassium may be increased for several reasons The pathology which causes *acute renal failure also causes destruction of tissue with* release of potassium Transfusions of banked blood often used early in treatment may constitute another source of potassium Furthermore if the patient is not vomiting orange juice and other citrus fruit juices which are sources of large amounts of potassium may be ingested Finally potassium is released in the process of normal protein catabolism

Cardiac Effects The most important effect of hyperpotassemia is on the conducting system of the heart and on the myocardium It may be anticipated by serial ECGs taken from the onset of anuria Tall peaked bizarre T waves are the earliest change this may be followed by prolongation of the *Q T QRS* and *P R* intervals The *P* and *R waves then* become smaller the RST segments depressed and the S waves larger Later the rhythm becomes grossly irregular due to premature beats and finally the ventricular complexes may appear as a continuous sine wave Death may be due to sinus nodal arrest or ventricular fibrillation

There is no specific correlation between the serum potassium level and the ECG changes An elevated serum *sodium*

antagonizes these changes and a lowered sodium or calcium level increases the characteristic changes of hyperpotassemia Since the heart is so responsive daily ECGs are required to discover such abnormalities It may be advisable in certain cases to correlate these findings with frequent blood determinations

Accumulated body potassium cannot be removed by the kidney Extrarenal routes of elimination must be sought two procedures may be used Potassium may be removed either from the gastrointestinal tract where it is present in high concentration or from the extracellular fluid Potassium is removed from the gastrointestinal tract by gastric or preferably by intestinal suction Catharsis in the constipated patient also drains the gastrointestinal tract of potassium Magnesium salts should be avoided because of possible aggravation of the existing hypermagnesemia Cation exchange resins also have been used to remove potassium from the gastrointestinal tract Most of these however contain potassium and are therefore contraindicated Resins of the hydrogen or ammonium cycle are available but their clinical application is so difficult that their use is limited to special instances

Potassium Antagonists The most powerful potassium antagonist is sodium When given intravenously sodium promptly reverses the clinical and sometimes the well advanced electrocardiographic manifestations of hyperkalemia Sodium is indicated where a rapid effect is required Two hundred cc of a 3 or 5% solution of the chloride bicarbonate or lactate is usually given The bicarbonate or lactate is preferred because transfer of potassium across the cell membrane

seems to occur more rapidly in the alkalinizing sodium This effect may be enhanced by adding the salts to a 5% glucose solution These solutions are infused rapidly the advantages, though rapid in action are not lasting and must be followed by more permanent therapy Furthermore sodium therapy may be a double edged sword although it reduces potassium levels the hazards in patients with hypertension and cardiac decompensation may be considerable Unless the situation is urgent other methods of treatment should be employed

Calcium is another potassium antagonist Hypocalcemia is a frequent finding in renal insufficiency The administration of calcium would therefore seem indicated Large amounts are needed for beneficial results 100 cc of 10% calcium gluconate may be used daily intravenously as prophylaxis against potassium intoxication It should of course be used cautiously in the digitalized patient

Glucose and insulin cause a temporary decrease in potassium levels because the deposition of glucose as glycogen in the liver requires the shift of potassium and phosphates to the liver The effect is temporary because potassium is released to the blood stream as glycogen is broken down However for a rapid decrease in the blood potassium level 200 cc of 25 or 50% glucose with 1 unit of insulin for each 2 grams of glucose is useful therapy

If the use of sodium calcium glucose and insulin fail to maintain the potassium below dangerous levels and symptoms and electrocardiographic changes persist one must resort to the use of the artificial kidney peritoneal irrigation or intestinal lavage These methods provide usually effective elimination of potassium from the blood

ACIDOSIS

Acidosis may be caused by loss of sodium or retention of acid metabolites As the acid metabolites accumulate the blood bicarbonate level must be lowered to maintain a normal blood pH This is accomplished by slow deep respirations (Kussmaul breathing) which "blow off" carbon dioxide Recognition of this phenomenon may influence the form of therapy Infusions of sodium will correct the acidosis due to sodium loss but may precipitate tetany if the acidosis is caused by retention of acid metabolites The preferred therapy for the removal of acid metabolites is the artificial kidney When this is not available 200 cc of 3 or 5% solutions of sodium bicarbonate or lactate may be used cautiously always keeping in mind the chance of tetany or pulmonary edema

Calcium and Phosphorus Since the hypocalcemia of renal failure is secondary to phosphate retention the administration of calcium salts is only temporarily beneficial to the patient with twitching or convulsions It may also be given but with due caution in patients receiving digitalis

Phosphate retention is reduced by a diet low in phosphate and by decreasing protein catabolism Aluminum hydroxide gels are useful as long term therapy to bind phosphate in the intestine This is more useful in the chronic than in the acute renal insufficiencies

ANEMIA

The most effective and practical therapy in the management of the nephritic anemias is the transfusion of freshly drawn

packed red blood cells banked blood should not be given because of its high potassium content The transfused cells survive but a short time and consequently the hematocrit returns to the pretransfusion level in a few days Therefore if the anemia is asymptomatic it is not advisable to transfuse unless the hematocrit is below 25

CONVULSIONS

Convulsions may be due to hypocalcemia *or* to hypertensive encephalopathy Hypocalcemic convulsions may respond to *calcium gluconate* or *lactate* given intravenously *In many* cases of uremia however convulsions are caused by hypertensive encephalopathy Here magnesium sulfate is the preferred medication because it decreases muscular irritability The adult dose is 0 5 to 1 gm to a total of 4 or 5 gms /day It may be given intramuscularly in a 50% solution or intravenously in a 3% solution The patient must be watched closely for respiratory depression Usually knee jerks become hypoactive before the muscles of respiration are affected if this occurs the infusion should be discontinued Alternative treatment consists of dilantin or barbiturates

The Heart in Acute Renal Insufficiency Death in acute renal failure is frequently due to congestive heart failure and pulmonary edema These may be prevented by avoiding overhydration Profound anemia may intensify the heart failure Hypertension especially of the diastolic type may aggravate heart failure and should therefore be treated with one of the hypotensive drugs Hexamethonium and hydralazine are preferred

Pulmonary edema is treated by the conventional methods

Oxygen aminophylline if bronchospasm is present phlebot omy if the hemoglobin level is not too low and digitalis as soon as congestive failure is evident A full digitalizing dose should not be given at any one time but small doses repeated until the desired effect is obtained Morphine must be used with caution because of possible nausea and vomiting

TREATMENT OF THE DIURETIC PHASE

The amount of fluid to be replaced should be determined by the state of hydration rather than by the urinary loss Occa sionally patients are overhydrated during the oliguric stage when diuresis occurs if the urinary volume is used as an index of the fluid allotment for the next day polyuria may be maintained On the other hand enough fluid must be given to prevent dehydration Two to three thousand cc is usually adequate

If acidosis occurs during diuresis it can be corrected with 40 120 mEq of sodium lactate daily There need be no hesitancy about the use of sodium because sodium is needed to compensate for that lost in the urine

With diuresis potassium may be lost and hypopotassemia may replace hyperpotassemia of the oliguric phase When the urine output is large there is no need to restrict dietary potassium In fact oral supplements of potassium salts may be needed Intravenous potassium is dangerous and should be reserved for those patients who cannot take food and are losing potassium rapidly Twenty mEq of potassium chloride diluted in 1 000 cc of water may be given intravenously slowly

Once the diuretic phase is well established food and fluid are permitted *ad lib* as long as the caloric intake is main

tained There is no reason to restrict protein intake because with improvement in renal function urea nitrogen will return to normal

Infections should be treated with the proper antibiotics Anemia may persist for some time after diuresis has begun Iron liver folic acid are of no benefit The anemia may correct itself or transfusion may be necessary

TREATMENT OF CHRONIC RENAL FAILURE

Although complete restoration of kidney function is not possible in the patient with persistent renal failure he may be helped to lead a more productive and comfortable life Renal function may become stabilized by treatment of intercurrent disease and by regulation of water electrolyte and protein metabolism The chief therapeutic objective is maintenance of the chemical composition and volume of the body fluids at normal levels This is done by imposing upon the kidneys such conditions as will compensate for failure of the kidneys to perform their function

Fluid Balance Thirty five grams of solids must be excreted daily by the normal kidneys to prevent azotemia Each gram requires 15 cc of water for solution Because the failing kidney cannot concentrate a daily volume of 2000 to 3000 cc is required to the 35 gms of solids Urine output greater than this may become less and less effective

Edema usually does not occur in uncomplicated renal insufficiency When it does occur it is due either to congestive heart failure for which digitalis is indicated to the nephrotic state which occasionally responds to cortisone, to an acute exacerbation of chronic *glomerulonephritis* or

it may be that the urinary output was unable to keep pace with the intake This might occur when the glomerular filtration rate is greatly diminished Forcing fluids here is contraindicated, water should be restricted just as it is in acute renal failure

Diet Between 2000 and 3000 calories are required to meet daily energy requirements There has been much academic controversy about the protein allowance in this diet Some authors favor 60 to 90 gms daily unless a rise in the urea nitrogen indicates that the patient cannot handle this amount When urea nitrogen rises protein is further restricted On the other hand the group led by Addis prescribes diets of 0 5 gm or less per day per kilo of ideal body weight plus an amount corresponding to that lost in the urine the preceding day Addis has presented calculations indicating that the excretion of urea requires more "work by the kidney than the excretion of other solutes To spare the damaged kidney this additional "work the protein intake should be sharply restricted

Both diets seem to be beneficial Generally the patient with mild or moderate urea retention is allowed unrestricted protein intake If there is a rise in urea nitrogen protein is restricted to 60 to 90 gms If renal insufficiency becomes severe further restriction to about 40 gms or less is indicated since the kidneys are unable to excrete adequately the metabolites resulting from protein destruction

Electrolytes Electrolytes may be excreted or retained and may produce a variety of patterns A given pattern may be relatively stable or may vary from time to time

Early in renal failure tubular reabsorption is impaired and sodium is lost The resulting hyponatremia may cause a decrease in glomerular filtration rate with retention of sodium Phosphate and urea are also retained and the uremic process is aggravated To avoid this it is important to prevent hyponatremia Sodium should not be restricted except in those who have hypertension or congestive heart failure The hyponatremia of chronic renal failure is treated similarly to that of acute renal failure

Acute potassium intoxication is rare in chronic renal failure so long as urinary output is adequate The blood potassium levels may be decreased in patients with polyuria but when polyuria is no longer a problem there may be a slight elevation of the potassium levels This has been attributed to a shift of potassium from intra to extracellular spaces If the urinary output is adequate and there are no electrocardiographic changes potassium salts may be given cautiously to avoid further shift from the cells and depletion

Phosphate and Calcium Phosphate retention is usually accompanied by hypocalcemia and often compensatory hyperparathyroidism This together with the acidosis generally present tends to make more ionized calcium available and thus prevents tetany Tetany is more likely to occur if an excessive amount of sodium has been given Treatment of hyperphosphatemia and hypocalcemia consists of the administration of a diet containing 0 5 gm or less of phosphate The nonabsorbable aluminum gels are useful in preventing *the absorption of phosphate by the gastrointestinal tract*

In addition appropriate administration of extra sodium to meet renal needs will spare calcium

Infections An acute exacerbation of chronic glomerulonephritis following a hemolytic streptococcal infection may add to the existing impairment of renal function For this reason prophylactic penicillin 200 000 u orally 4 times a day should be given for all respiratory infections However the evidence that exacerbations can be prevented in this way is not entirely convincing

Hypertension The greater and the more fixed hypertension becomes the poorer the outlook is likely to be Treatment becomes more difficult as renal impairment advances Sodium restriction may embarrass the renal circulation and may increase the depth of the uremia Sympathectomy is contraindicated because the patient is usually a poor operative risk Adrenalectomy is also contraindicated because of the electrolyte changes which follow such a procedure Hexamethonium and hydralizine have sometimes been helpful temporary improvement in congestive heart failure and pulmonary edema have occurred in well advanced renal disease This form of treatment is potentially dangerous however because a drop in blood pressure may aggravate renal failure It might be well to reserve this form of therapy for those whose specific gravity is not fixed and who have reasonably good function as measured by the PSP and urea clearance tests

Gastrointestinal Symptoms Anorexia nausea and vomiting are troublesome symptoms Small frequent feedings may

control the nausea If this fails Thorazine and Compazine may be used with frequently gratifying results Compazine is preferred because it is less toxic It may be given orally in doses of 10 to 25 mg 3 times a day or intramuscularly in doses of 5 or 10 mg A rectal suppository containing 25 mg of the drug is also available

Peptic ulcer is not an uncommon complication It requires the usual treatment except that drugs eliminated slowly by the kidney are to be avoided

Skin Complications Scrupulous care is required to avoid infections Pruritus often responds to antihistamines but these should be used cautiously because of possible drowsiness and urinary retention Compazine occasionally relieves the itching

ARTIFICIAL KIDNEY

The first artificial kidney was devised in 1903 In this apparatus crystalloids diffused through the pores of a collodion membrane into a rinsing fluid This method was impractical and discarded

In 1938 commercially available cellophane sausage casing was suggested for use as the semipermeable membrane Five years later Kolff constructed the first clinically successful artificial kidney utilizing heparin as the anticoagulant Since then many other types have been devised The mechanism of all of the artificial kidneys depends upon the principle of dialysis with blood on one side of the membrane and the rinsing fluid on the other

The cellophane casing is a cellulose membrane containing glycerol for softening and a small amount of water Boiling

and rinsing in tap water swells the membrane It then contains submicroscopic capillary channels with a pore radius about that of the glomerular capillaries The size of the pores is such that large molecules and blood cells are retained but crystalloids urea uric acid creatinine and phenols may pass through Such diffusion is known as dialysis

The cellophane tubing is wound around a drum and attached at both ends to fittings which lead to rotating couplings The drum is partly submerged in a bath solution Blood from an artery is forced through the apparatus and a pump returns it to a vein Heparin is used as an anticoagulant

A more recently developed disposable artificial kidney has the advantage of requiring less preparation It consists of twin coils of cellulose tubing separated by fiberglas screens sewed together and wound on a stationary coil A larger dialyzing surface area possible with this design increases the rate of urea clearance The entire apparatus fits into a metal can and is ready for use after having been washed with saline

The bath fluid contains the essential electrolytes in concentrations similar to normal plasma Lactic acid is added to bring the pH to 7 4 It is maintained at this level by a constant stream of 10 per cent CO in oxygen The addition of glucose 250 mg per cent or more makes the solution hypermolar and supplies calories The composition may be changed according to the need for the removal of any specific substance For example if potassium is to be removed from the blood this is omitted from the bath fluid Back diffusion is prevented by using large volumes of bath fluid or changing it frequently Electrolyte equilibrium between the blood and rinsing fluid can be obtained by continued dialysis

The previous occurring complications like hemolysis clotting pyrogenic reactions and hemorrhage have been practically eliminated Severe hypertension is frequent when the blood flow through the artificial kidney is 200 cc or more per minute It can be reduced quickly by hexamethonium Advantage has been taken of the rise in blood pressure in using the artificial kidney for the hypotension of severe shock

Use of the artificial kidney requires a trained group not only experienced in the use of the apparatus but with a knowledge of the physiology of electrolytes and body fluids

If the equipment is not available intestinal lavage or peritoneal lavage may be used By using the mucosa as a dialyzing membrane moderate amounts of urea can be removed but with little change in the blood level The process is complicated by the fact that the intestinal mucosa secretes and absorbs electrolytes independently of the concentration differences between the intraluminal fluids and the blood The method is limited by the difficulty in properly placing the tube and by the fact that retention products other than urea diffuse poorly

Peritoneal dialysis is based on the concept that the peritoneum can act as a semipermeable membrane There are different techniques some using continuous and some intermittent lavage through an inflow and an outflow catheter Urea creatinine and other retention products can be removed in significant quantities The composition of the rinsing fluid is similar to that used in the artificial kidney

INDICATIONS FOR THE USE OF THE ARTIFICIAL KIDNEY

Because of the many limitations and difficulties encountered in the use of intestinal and peritoneal lavage the artificial

kidney is at this time the preferred method for removal of retention products

Any patient with acute or chronic uremia may require the artificial kidney at some time to tide him over an emergency The indications for its use vary and depend to some extent upon the availability of the apparatus and the necessary personnel When these are ready the indications for dialysis are extended If the apparatus and the team are not so easily available the use of the artificial kidney is limited to those whose blood urea nitrogen is between 150 and 200 CO combining power 12 or less and potassium 7 mEq/liter or more These are indisputable indications There are many cases which fall between the early uremias and those with severe manifestations In these clinical judgment will determine whether or not dialysis is indicated

1 *Acute Glomerulonephritis* Convulsions have occurred during dialysis in the early stages Treatment should be reserved for the progressively severe uremias

2 *Acute Tubular Necrosis* If the patient is asymptomatic and shows evidence of a spontaneous recovery with conservative measures dialysis is unnecessary However dialysis is indicated if a rapid rise of the urea nitrogen and an increase in the electrolyte imbalance occurs in spite of conservative therapy Dialysis should also be used in those who fail to diurese by the seventh or the tenth day or deteriorate after diuresis has occurred In these dialysis should not be delayed but should start early before irreversible changes have taken place

3 *Chemical Poisonings* Those which are nephrotoxic include bichloride of mercury carbon tetrachloride potassium dichromate potassium chlorate sodium potassium tartrate mushroom poisoning and sulfonamide poisoning

Salicylates and bromides which have no effect on the kidney are readily dialyzable Barbiturates vary in their ability to pass through the dialyzing membrane Those which are bound to the plasma proteins (secobarbital and amybarbital) do not dialyze Pentobarbital passes through the membrane poorly but phenobarbital can readily be removed from the blood by the artificial kidney

4 *An Acute Exacerbation of Chronic Uremia* This is treated like acute uremia with the artificial kidney

5 *Intractable Edema* as a complication of acute or chronic intractable edema uremia If the rinsing fluid is made hyperosmolar with glucose a large volume of edema fluid can be removed

6 *Dialysis* This may be used as a preoperative measure in the uremic patient who requires surgery

Contraindications Active bleeding was considered the only contraindication to the use of the artificial kidney but recent experience has shown that such cases may benefit

ACUTE RENAL FAILURE

Acute renal failure has been variously known as acute tubular necrosis lower nephron nephrosis crush syndrome hemo-

globinuric nephrosis and acute necrotizing nephrosis The different designations were meant to stress either the cause of the syndrome or the physiologic or structural changes Whatever the cause decreased circulation through the kidney due to the hypotension of the initial shock and vasoconstriction is important in initiating and maintaining an insufficient blood flow It is possible that substances elaborated by the injured part play a role A variety of precipitating factors have been identified These fall into several categories

1 *Decrease in Renal Flow* The most important are those conditions which cause a shocklike state and decrease the renal blood flow These include extensive trauma hemorrhage severe fluid loss infections or hypoxia

2 *Free Hemoglobin* Caused by a transfusion accident free hemoglobin in the plasma may result in *hemoglobinuric nephrosis* Other infrequent causes may be intravascular hemolysis following introduction of distilled water into the blood stream copper sulphate intoxication eclampsia and hemolysis due to infectious organisms

3 *Toxins* which damage specific parts of the renal tubule produce *necrotizing nephrosis* Among the agents causing such injury are bichloride of mercury carbon tetrachloride and mushroom poisoning

4 *The Hepato Renal Syndrome* This may vary from the mild cholangitis associated with acute glomerulonephritis to the severe acute hepatitis with acute renal tubular necrosis How the liver causes renal damage is not known

CLINICAL PICTURE

Rapidly progressing impairment of renal function is the outstanding feature An early abrupt fall in urinary output which may progress to anuria usually lasts 10 to 12 days though it may persist for weeks this is the oliguric phase The blood pressure normal at the onset soon reaches hypertensive levels Edema is not a characteristic feature its presence suggests overhydration Severe renal insufficiency often results in uremia with a low grade fever vomiting diarrhea convulsions delirium and pulmonary edema However, the major problems at this time are not related to the renal insufficiency They are rather cardiac complications Vigorous hydration may lead to fatal pulmonary edema Potassium retention may cause hypotension bradycardia the characteristic arrhythmias and finally asystole

The oliguric phase is ended when the urine volume reaches 1000 cc 24 hour period It is followed by the diuretic phase The tubular epithelium is sufficiently restored to block reabsorption but it cannot concentrate the glomerular filtrate Consequently the plasma may become depleted by electrolytes The urea nitrogen usually remains elevated and may even rise until the output reaches 3000 cc The symptoms of renal insufficiency often persist until diuresis is well established Marked anemia and inability to concentrate may last for weeks or months after clinical recovery

Laboratory Findings A smoky brown or red urine suggests acute tubular necrosis In other instances of acute renal failure the urine is not characteristic in appearance The specific gravity of the urine is low from the onset except in acute

tubular nephrosis where it is high for a short time Albuminuria is constant The sediment contains casts and red blood cells Pigmented casts, either hemo or myoglobin are characteristic of acute tubular nephrosis Leucocytosis and a progressive anemia are constant findings

Continued renal insufficiency leads to azotemia and uremia with its retention products and electrolyte disturbances

Prognosis The prognosis depends to a great extent upon the lapse of time before treatment is instituted During the oliguric phase uremia mild or severe and the cardiac complications are serious The prognosis should be guarded even after the onset of diuresis It is only after diuresis is well established that optimism is warranted After clinical recovery the specific gravity may remain low for many months and the anemia may persist after all signs of uremia have cleared Clearance tests may be impaired for years but eventual functional recovery can be expected

Treatment Other than the specific treatment for the causative agent the treatment of acute renal failure is presented in the section of acute uremia

TOXEMIA OF PREGNANCY

Toxemia of pregnancy is an acute vascular disorder characterized by *albuminuria* without other manifest cause generalized *edema* and rise in *blood pressure* above the prepregnancy levels These symptoms subside with the termination of the pregnancy Severe toxemia may progress to eclampsia a form of hypertensive encephalopathy

Symptoms Toxemia occurs during the second half of pregnancy The symptoms consist of edema headache nausea vomiting scotomata blurred vision drowsiness coma and convulsions *Systolic and diastolic hypertension, generalized* edema and proteinuria are dominant features The eye grounds are normal in mild cases but in advanced cases arteriolar spasm papilledema and retinal detachment are likely In mild toxemia the heart is normal later signs of cardiac insufficiency appear Progression of the symptoms results in coma and convulsions oliguria or anuria The syndrome may eventuate in pulmonary edema cerebral edema or renal failure

Diagnosis Hypertension in pregnancy may be an expression of preexisting hypertension or it may be due to toxemia Toxemia may appear in those who were normotensive before pregnancy or it may be superimposed upon a previously elevated blood pressure The differentiation can best be made in retrospect

Those with normal blood pressure *usually exhibit* a tendency to hypotension during pregnancy Any elevation of blood pressure and/or albuminuria during the second half of pregnancy in a previously normotensive woman suggests toxemia These signs clear fairly rapidly after delivery Patients with prepregnancy hypertension may go through pregnancy without any change in blood pressure and/or albuminuria However some of these prepregnant hypertensions may have *further elevation of blood pressure* and increase in albuminuria *during the second half of pregnancy* In these toxemia is superimposed on the prepregnant hypertension Again,

these signs are usually reversed several weeks or months following the termination of pregnancy

Laboratory Findings Albuminuria is a fairly constant finding Microscopic red blood cells are rare except in eclampsia Casts are common

Urea clearance tests are equivocal Renal blood flow is normal but glomerular filtration may be decreased

ACUTE PYELONEPHRITIS

Acute pyelonephritis may be initiated by a hematogenous infection an ascending infection or obstruction in the urinary tract It is ushered in by chills fever urgent urination dysuria low back pain and costovertebral angle tenderness Pyuria and bacilluria may be intermittent The process is essentially an inflammation of the interstitial tissue In the acute stage renal tissue is not destroyed to any appreciable extent therefore renal function pyelography and blood pressure are normal Acute pyelonephritis may heal completely it may recur it may progress to acute necrotizing papillitis or it may become chronic

Acute necrotizing papillitis occurs more commonly in diabetics Clinically the picture simulates that of simple acute pyelonephritis with hematuria oliguria anuria and azotemia The process may start as a fulminating septicemia it may be present as a severe exacerbation of acute pyelonephritis or rarely the course may be more benign in its progress to uremia Diagnosis depends upon the clinical picture of acute pyelonephritis the finding of sequestrated

papillary tissue in the urine and the characteristic destruction of the papillae on pyelography

Chronic Pyelonephritis If acute pyelonephritis becomes chronic, the gradual contraction of the interstitial tissue resulting from the inflammatory process leads to a gradual amputation of the functioning renal tissue with eventual renal insufficiency The same process may be responsible for renal vascular changes which are often associated with marked hypertension

Treatment Because of the occasional malignant outcome of acute pyelonephritis and its tendency to chronicity, treatment should be instituted early Measures are taken to relieve any existing obstruction and/or ascending infection The urine should be cultured Bacilli of the colon group are most frequently found staphylococci are less frequent and streptococci and proteus are rare The antibiotic to which the causative organism is sensitive is used with the object of *eradicating the infection* in the interstitial tissue rather than *sterilizing the urine* If the organism is sensitive to an antibiotic treatment is continued for ten days A resistant organism requires more prolonged treatment No patient is considered cured unless three successive urine cultures are negative after all traces of the therapeutic agent have disappeared from the urine Repeated urine cultures should be negative for six months

ACUTE GLOMERULONEPHRITIS

Clinical symptoms of acute glomerulonephritis usually follow ten days to four weeks after a Group A hemolytic

streptococcus infection The onset is characterized by fever headache anorexia vomiting and occasionally convulsions Outstanding features are edema urinary changes and a temporary rise in blood pressure

Edema may vary in extent Rarely it is latent The cause of the edema is not clear Some attribute it to capillary damage Others believe it due to congestive heart failure because enlargement of the heart pulmonary edema increased venous pressure and a palpable liver are frequent in the severely ill

Urine Oliguria occurs in practically all cases Suppression may amount to anuria Early the specific gravity is normal It may fall to hyposthenuric levels with diuresis or impairment of renal function Proteinuria is slight Hematuria either microscopic or gross is significant It may be transient or persist until healing is complete White blood cells and casts of all types are usual in the sediment

The blood pressure may or may not be increased If it rises it generally returns to normal as healing occurs Continued hypertension after clinical healing indicates that the process is becoming chronic Usually the eye grounds are normal Hemorrhages and/or exudates indicate a chronic process

The clinical course of acute glomerulonephritis varies Most recover completely in several weeks There is no relationship between the severity of the attack and the outcome In those who recover renal function improves but proteinuria hematuria and the inability to concentrate may persist for months In a small number who survive renal

function deteriorates steadily as they progress through the subacute to the chronic stage

As soon as the acute symptoms appear strict bed rest is enforced until the condition is stabilized Food consists of carbohydrates mainly Because of their high potassium content proteins and citrus fruits are omitted until diuresis begins The treatment of the oliguria and anuria and the *complications is described in the section on the treatment of acute renal failure*

Prophylaxis is impractical However if an outbreak of a nephrotogenic strain of streptococci occurs in a susceptible group penicillin should be used prophylactically

Part VI

BARBITURATE INTOXICATION

Jack Abry M D

XXXII BARBITURATE INTOXICATION

About seventy derivatives of malonylureas are employed in daily medical practice as sedatives and hypnotic agents The consumption in the United States of America is around three hundred tons yearly and is responsible for nearly fifteen hundred deaths each year In twenty five per cent of all cases in which patients are admitted to general hospitals for acute poisoning the barbiturates are the intoxicants The fatality rates range from eight to twenty five per cent depending on severity and timing of therapy However in certain small series the results are better and average 1 6 to 3 4 per cent of hospitalized cases

Many acute barbiturate poisonings arise from voluntary suicide sometimes the intoxication is unintentional and stems from accidental self poisoning while mentally confused from a previous tablet taken before retiring Concomitant alcohol consumption or oversensitivity to a particular barbiturate may also cause intoxication

CLASSIFICATION OF BARBITURATES

Barbiturates may be classified into four common groups

A Long acting (phenobarbital)
B Intermediate duration of action (Amobarbital and Amytal)
C Short acting (Pentobarbital and Nembutal)
D Ultrashort acting (Thiopentone Sodium or Pentothal)

Although these drugs vary in human toxicity ten to fifteen times the average dose or about 1 gram usually produces serious intoxication in an adult Death is likely to occur if the intake is greater than two grams Fatalities usually develop on the second to fifth day after ingestion and prognosis may be favorable if the patient survives the first thirty six hours Aggravating factors are old age liver pathology and cardiovascular disabilities Obesity associated with other toxic syndromes chronic alcoholism opiates or hypersensitivity to the barbiturates are also important factors The basic pathology and clinical picture of barbiturate poisoning is one of progressive central nervous system depression The most important clinical aspects of the syndrome are respiratory and circulatory complications

CLASSIFICATION OF INTOXICATION

Barbiturate poisoning can be classified clinically as mild moderate and severe depending upon the degree of the central nervous system depression Moderate poisoning is characterized by impairment of mental ability and judgment, emotional instability garrulousness and drowsiness However sometimes other symptoms like transient nystag

mus occur but in general reflexes other than superficial twitches are not depressed Respiration and blood pressure are not significantly changed Such patients may be aroused by physical stimulation

An individual under barbiturate overdosage looks like a patient in deep anesthesia the condition also resembles profound alcoholic intoxication

Coma Severe poisoning is characterized by deep coma from which the patient cannot be aroused by any type of stimulation A clammy cold skin covered by perspiration more or less constant nystagmus disappearance of tendon reflexes (positive Babinski) constricted pupils (dilatation occurs when hypoxia develops) absent corneal and pharyngeal reflexes marked depression of respiration which later becomes rapid and shallow or slow and labored and sometimes Cheyne Stokes or Kussmaul breathing occurs Further symptoms are cardiovascular collapse manifested by a rapid weak pulse hypotension (an alarming symptom when systolic blood pressure falls below 65 mm of mercury) and cyanosis There may be a relative grade of urinary suppression secondary to renal failure Death may occur by respiratory arrest acute pulmonary edema or circulatory collapse If the patient survives longer than five to ten days fatalities may occur from hypostatic or aspiration pneumonia and also from lower nephron nephrosis with uremia

Differential Diagnosis Fortunately only patients with severe barbiturate intoxication require intensive medical attention and treatment The diagnosis of dangerous barbiturate poisoning is not always a simple procedure A positive

history of ingestion of a large amount of the drug obtained either from the patient's relatives or friends is important Medicine bottles, capsules or tablets found near the patient offer a clue Characteristic colors of certain capsules (nembutal—yellow) imparted to the mucous membranes of the mouth or in the gastric washing may be informative When such data are lacking the examiner must consider other etiologic factors responsible for clouding of the sensorium and coma Head injuries cerebro vascular accidents brain tumors meningitis and encephalitis coma of diabetic uremic and hepatic origin, cardiovascular accidents poisoning with methyl alcohol, morphine bromides and other drugs must be kept in mind Laboratory tests in which chemical identification of the barbiturate preparation found in the gastric contents blood or urine as well as the characteristic electroencephalographic pattern of the comatose patient confirm the diagnosis in hospitalized cases

TREATMENT

After establishing the diagnosis first aid should be started before rushing the patient to a hospital Certain first aid rules may be mentioned here First attention should be paid to the patient's airways residual contents of the mouth like food or portions of the drug itself must be removed Dentures should not be forgotten Secretions from the trachea and bronchi must be aspirated Secondly note patient's respiration if shallow or absent start artificial respiration promptly using the mouth to mouth procedure if possible If complete or partial airway obstruction has occurred tracheotomy must be considered and performed without delay

Thirdly note patient's blood pressure if at systolic levels

below 65 mm Hg emergency analeptics should be administered Caffeine Coramine Wyamine may be given intramuscularly epinephrine preparations may be dangerous

Fourthly maintain body heat by appropriate garments or coverings heat loss during the unconscious or coma state is said to be about three to four times that of the normal individual

On admission to the hospital prompt attention should be given to gastric lavage it is doubtful that this procedure can be performed safely or adequately at home or under similar circumstances

Gastric lavage is not always a simple procedure and it should not be left in the hands of untrained personnel The type of gastric tube its size the characteristics of the bulb *end the degree of suction to be applied the volume of wash* water its temperature and composition all may have an important bearing on success or failure in any given case The danger of perforation either of the lower end of the esophagus or of the stomach wall is always present The first aspirated contents should be sent to the laboratory for prompt analysis Experience has shown that tap water at body temperature produces the least shock and complications magnesium sulphate alkalies like bicarbonate of soda acids like vinegar or the aluminum gel preparations are contraindicated While the lavage tubing may be inserted more or less chilled cold water enhances the hypothermic symptoms of shock hot water on the other hand may increase the edema of all mucous membranes At least five to six liters of water should be used after the first washing which should be performed quickly subsequent lavage may be made slowly Except in very toxic patients it is not uncommon to witness

some signs of returning consciousness after the first half hour of treatment in the emergency room At this time there may be a slight or partial return of the gagging and swallowing reflexes

Intravenous Therapy The question of intravenous medication requires serious consideration if the patient is known to have been unconscious for a period longer than 5 to 6 hours dehydration has probably taken place the urinary bladder should be catheterized If no or little urine is obtained renal involvement must be suspected here the usual methods of treating kidney function arrest must be promptly instituted The lower nephron syndrome should be suspected and therapy based upon the concepts discussed in a previous chapter should be instituted If urine can be removed from the bladder prompt analysis is indicated to determine the presence of proteinurea and red blood cells Sugar is not infrequently discovered but this finding of glycosuria should not be confused with diabetes a blood sugar determination is important here Most instances of barbiturate coma show a relative hypoglycemia the employment of intravenous glucose solutions in such instances represents effective supportive therapy

Cardiac Effects Unless the patient has had previous cardiovascular disability no special problems arise in regard to the heart the rate is usually slow due to vagal effects produced by the drug The rate rarely goes below 52 the rate is regular but occasionally premature beats are noted The electrocardiogram may show some changes in the ST segments in Leads 1 2 AVR and the axillary V leads rarely there are

conduction disturbances with a lengthening of the P R interval to about 0 12 seconds A few instances of QRS delay have been recorded In a large series of cases seen at New York City Hospital no instance of coronary involvement was seen notwithstanding the low blood pressure levels

Shock Treatment After the emergency procedures have been performed the patient should be placed under constant nursing observation The bed should be in reversed Trendelenberg position with the legs elevated The patient should be placed in shock position with the head at least twelve to fourteen inches below the legs but it should be noted that while this may be the optimum position to maintain a competent cerebral circulation the gravitational effect upon the lung field may enhance the likelihood of hypostatic or congestive pneumonia All patients in coma require prompt antibiotic therapy the hazard of inspiratory pneumonia is always present and the threat of staphylococcus infection has become an increasing problem in recent months

Drug Therapy Unfortunately there has not been developed specific antidotal treatment for barbiturate overdosage nor have chemical neutralizers been found to be effective With the basic pathology chiefly involving the central nervous system successful therapy has been based upon attempts to stimulate the respiratory center Drugs like picrotoxin metrazol strychnine ephedrine amphetamins and similar central nervous system stimulants do not neutralize the toxic effects of the barbiturates but artificially stimulate nervous centers already damaged this has been compared to the whipping of a tired horse It may temporarily succeed but more often

these drugs enhance the symptomatology of the pre existing pathology

On the other hand treatment after the initial gastric lavage *is chiefly symptomatic and supportive and will vary from pa*tient to patient

Symptomatic Therapy Therapy here is similar to that employed in coma due to other causes The objective being to maintain such vital physiology as respiration circulation and excretory functions at their optimal state until the toxin is excreted or metabolized Of primary importance is continued airway hygiene Suction of tracheo bronchial secretion or the use of indwelling oral pharyngeal catheters is important If the patient is in profound coma for eight to ten hours an endotracheal tube for removing the inspissated bronchial secretions is mandatory Atropine in 0 6 mg subcutaneously twice daily is indicated to lessen bronchial spasm and hyper secretions If breathing is shallow mechanical respirators are advisable Sometimes inadequte pulmonary *ventilation can* be improved by cautious use of analeptic drugs The most effective are caffeine (0 25 to 1 0 gram subcutaneously intra muscularly or intravenously every three hours) niketamide (coramine) 1 to 3 ml by any route alternating with caffeine or other analeptics like ephedrine (hydrochloride or sul phate) 30 to 50 milligrams every half to two hours or am phetamine by any route 10 to 30 milligrams repeated every 30 to 120 minutes as necessary These drugs must be admin istered with special attention for their side effects Sometimes excessive therapy may be harmful and should be avoided

The threat of cardiovascular collapse usually disappears when and if hypoxia is corrected The above mentioned

analeptics should also be used if cardiovascular collapse ap pears to be imminent especially when blood pressure drops to hazardous levels (systolic below 65 mm Hg) In such cases Levophed (Levarterenol) should be used (4 cc of Levophed and 1000 cc of 5 per cent glucose intravenously at the rate of 10 to 20 drops per minute) Sometimes a plasma expander (dextran) or transfusion of whole blood or plasma may lessen the danger of severe circulatory collapse

Intravenous Medication Dehydration may be corrected by cautious use of replacement fluids (2 to 3000 cc daily glu cose 5 per cent or normal saline solution) Hypertonic saline solutions have been used in certain selected patients where urinary suppression has already occurred the concept of Parsonnet concerning the stimulating effects of increased NaCl concentration in the renal arterial circulation may en hance glomerular activity in patients suffering from critically low systolic blood pressure levels Signs of pulmonary con gestion and pulmonary edema should be noted Fluid re placement if extensive or long continued may be accompa nied by serious disturbances of electrolyte balance which if unrecognized may be productive of a number of complica tions the most serious of which are the cardiac arrhythmias The prophylactic use of the potassium preparations should be considered provided there is an adequate urinary output

Fluid Balance A daily balance of fluid intake and output has some clinical importance in certain patients barbiturate overdosage is associated with a specific type of urinary sup pression It is possible that an antidiuretic hormone substance related to secondary hypothalamic or pituitary reactions is

produced in the general central nervous system involvement Catheterization is frequently necessary and an indwelling catheter should be employed

A second gastric lavage may be indicated after the first twenty four hour period there is some evidence that certain breakdown products in barbiturate intoxication may be excreted into the stomach and unless removed, may delay recovery Vomiting may occur in patients who have shown signs of returning consciousness this vomiting may be central but more often it results from these excreted esters of the malonylureas the basic compound of the barbiturates Experiments have shown that after tap water lavage potassium permanganate in 1 to 5000 solution may have a neutralizing effect upon these substances Sodium sulphate in concentrated solution (from 20 to 30 cc) may also be effective both by its physiologic neutralizing action as well as from its cathartic possibilities

Electrical Stimulation The marked depressive syndrome seen in barbiturate intoxication has been compared to lesser depressive states noted in the practice of psychiatry in 1950 Markham suggested that electroshock therapy might have a field of usefulness in drug induced depressions Where such electroshock apparatus is available like the Reiter modulated unidirectional periodic electrocerebral stimulator currents up to ten milliamperes may be applied in the usual manner to the head of the patient Individuals who respond show increased ventilation as well as improvement in circulation and reflexes Patients in mild and moderate coma are much more responsive to this treatment than the profoundly unconscious

Treatment of the Preagonal State When the above mentioned measures have failed to counteract the overall depression of the central nervous system the use of certain potent and controversial agents must be considered Among these are Metrazol and Picrotoxin These drugs are general central nervous system excitants starting with the cortex and involving the vital centers of the spinal bulb Picrotoxin first introduced in 1935 by *Arnett has demonstrated its spectacular* effect in many apparently hopeless cases of barbiturate poisoning Its demonstrated action on the brainstem improves respiration and circulation and hastens the reappearance of both the deglutition and cough reflexes By inducing a certain amount of restlessness and active movements on the part *of the patient it prevents dangerous circulatory stasis* Given however more often than thirty minutes bv the intravenous route it may produce dangerous convulsions and fatal hypoxia

Metrazol and Picrotoxin Metrazol (Phenylterrazol) though more immediate and transitory in its action than Picrotoxin is more effective on the higher central centers It must be used in appropriate dosage in order to avoid convulsions Some clinicians prefer to start with Metrazol because Picrotoxin is more likely to produce these convulsions but others prefer Picrotoxin because of this very effect Controversy here stems from differential responsiveness of patients to these two drugs Nielsen (1951) who reported a series of 176 cases treated by conservative measures at a barbiturate poisoning center showed a low fatality rate of three to four per cent This compares very favorably with other reported series where potent analeptics like Metrazol and Picrotoxin were

used According to Neilsen the amount of barbiturates ingested is not as important as is its specific effect upon circulatory and respiratory function This is in direct contradiction with the generally accepted rule that the depth of the coma and the effect of the barbiturate on the respiratory and circulatory centers is directly correlated with the amount of poison ingested and retained

Another objection of the Scandinavian author is that potent central acting analeptics may cause serious secondary and irreversible depression of the central nervous system Animal experiments show that convulsive doses of Picrotoxin produce a marked increase in cerebral oxygen utilization Indicated dosage of Picrotoxin and Metrazol should be carefully administered a few rules may be mentioned First start with 5 cc of 10 per cent solution of Metrazol given intravenously slowly If twitching and grimacing appear in addition to return of corneal and deep reflexes Metrazol is the treatment of choice and the same dosage should be injected intravenously every 15 to 30 minutes until the patient is out of imminent danger Second if no appreciable response occurs the administration of 3 cc of the same solution at the same rate should be repeated at 15 minute intervals The dose should be increased on each succeeding injection until a maximum of 5 cc has been given Some clinicians prefer the slow intravenous route with 1 cc (3 milligrams per minute) until muscular twitches and reflexes reappear As much as 1000 to 2000 milligrams of Picrotoxin have been given (10 to 20 times the lethal dose) over a period of 2 to 5 days with life saving results

When the degree of coma lessens the patient should be stimulated to the point of being constantly awake but such

activity should be short of the production of convulsions The threat of central nervous system hypoxia is always present during a convulsive period It may require a fine sense of judgment and experience to regulate dosage and activity in such instances

Other drugs have recently been used to neutralize barbiturate intoxication Disodium Succinate has shown some promise but the drug has not yet been widely accepted The general consensus is that this type of therapy has not given acceptable evidence of definite benefit

Another substance Megimide (BB Methylethylglotarimide) has a stimulating action on the central nervous system similar to—but less intense than—Picrotoxin It is available as an experimental drug

Hemodialysis Of considerable clinical interest is the increasing use of the so called artificial kidney in instances of barbiturate intoxication accompanied by marked renal involvement Such patients usually present a history of long standing renal disability occasionally with azotemia and rarely with previous episodes of uremia Urinary suppression and lower nephron functional incapacity may develop early in barbiturate overdosage either by the mechanism of subcritical systolic blood pressure levels or by responsiveness to the antidiuretic humoral factors Here hemodialysis may be life saving in bridging the dangerous period of acute renal failure

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